THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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FRAGARIA BRACTEATA HELLER AS AN INDICATOR PLANT OF STRAWBERRY VIRUSES

Norman W. Frazier

It is a well-established fact that strawberry species and varieties may differ markedly in their reaction to strawberry virus diseases. Investigations of strawberry viruses are greatly enhanced by the use of an indicator plant that may readily be infected with the different viruses and will clearly express symptoms of the diseases they cause.

Harris and King (2) found the woodland strawberry Fragaria vesca L. to be the most useful of several species tested as possible indicator plants of strawberry viruses and this plant has subsequently been extensively used in strawberry virus investigations in Great Britain and elsewhere. Demaree (1) reported F. vesca to be a much more suitable indicator plant for strawberry yellows disease than the commercially grown American variety Marshall.

An investigation of the insect transmission of strawberry viruses was undertaken during 1939 by the author working with Harold E. Thomas. Early work proved the variety Marshall to be quite unsatisfactory as a test plant. A reliably virus-free source of Marshall was not available and a smaller, more readily handled test plant was desired. Tests were made both by grafting and by insect transmission of the susceptibility to virus infection of a wild strawberry determined at the University of California Herbarium to be the bracted strawberry, Fragaria bracteata Heller. This species appeared to be an excellent test plant that showed marked sensitivity to infection, good differentiation of symptoms, and suitable adaptation to year-round greenhouse conditions. F. bracteata was therefore used as a standard indicator plant in all subsequent work.

The investigations were suspended during 1941 and after resumption again in 1949 a number of other species or varieties of wild Fragaria have been compared with F. bracteata for their possible usefulness in strawberry virus work. Two varieties appear to have considerable value and are now being routinely used as indicator plants with F. bracteata. One is the East Malling strain of F. vesca and the other is a runnerless type of an Alpine variety of F. vesca. The sensitivity to infection and type of symptom reaction to the few viruses (as yet unidentified) used has been somewhat variable between the three hosts. For example, in the case of one virus the symptoms expressed by F. bracteata are much less severe than are expressed by the others, while in the case of a second virus the symptoms produced in F. bracteata and F. vesca are somewhat obscure and unreliable but in the Alpine variety are very distinct and superficially of a different type.

In order to broadly survey the possible viruses to be encountered in commercial strawberry plantings and at the same time to obtain information on the reactions to them of F. bracteata, two types of preliminary tests have been carried out.

One type of test has involved the direct transfer to healthy indicators of strawberry aphids, Capitophorus fragaefolii Ckll., found naturally infesting diseased commercial plants in the field. The number of aphids transferred to a single test plant varied from 1 to 10 and the length of the test feeding period varied from 6 to more than 15 days. Forty-seven of 75 inoculated indicator plants developed symptoms of virus infection. Fourteen infected plants displayed chlorotic, stunted, yellows type symptoms after an incubation period of one month or more unaccompanied by mottling, marginal chlorosis, or leaflet cupping. Thirty -three infected plants developed symptoms of the crinkle type, after one to three weeks, which varied considerably both in type as well as severity of expression, in many instances being rapidly lethal. Two general types of crinkle appeared to be distinguishable: a mild type consisting of typical mosaic-like mottling accompanied by little or no leaf malformation and only moderate stunting and dwarfing, and a more severe type with leaf malformation, chlorotic spotting and mottling, stunting, dwarfing, crown proliferation and often very early death. There appeared to be intergradation between the two types. Sometimes both the yellows and crinkle types of disease were produced by different single aphids removed from a single leaf of the inoculum plant.

The second type of test consisted of placing non-infective stock colony strawberry aphids on diseased field plants which had been potted and fumigated. The aphids were allowed various acquisition feeding periods of from 2 to 10 days, then divided into lots of 10 aphids and each lot transferred daily to successive healthy F. bracteata plants until all aphids had died.

Twenty-seven lots of aphids were transferred from 10 inoculum plants which included typical examples of yellows and crinkle as well as others which were of atypical appearance. All test plants inoculated on the first transfer from the inoculum plants developed disease of the crinkle type; all 5 plants inoculated by aphids from typical yellows plants developed the least severe symptoms, whereas 22 plants inoculated by aphids from typical crinkle or the other atypical

source plants developed very severe crinkle symptoms of which 17 were of the rapidly lethal type.

Infections of the yellows type only were produced irregularly beginning with the second set of indicator plants and continuing until the fifteenth set when the last infection in these tests took place. In all, 66 of 270 inoculated indicator plants became infected with yellows. The yellows diseases were not all identical but varied in severity and in the presence of a type of chlorotic spotting in some plants. Some of the variation could be correlated with individual inoculum plants.

It seems very probable from the observed variations and intergradations, that both the crinkle and yellows types of disease may be caused by more than one virus, or by a complex of viruses.

A third basic type of symptom on F. bracteata that has appeared on a few occasions consists of a drooping or downward arching, umbrella fashion, of the midrib towards the petiole. The midrib often lies at the bottom of a shallow trough formed by a slight inward folding of the leaflet margins. The severity of symptoms varies markedly and may be accompanied by a considerable degree of stunting. It appears probable that this droop type of disease likewise involves more than one virus.

On the basis of these preliminary trials with F. bracteata indicator plants it appears that the strawberry viruses most common in California might be broadly separated into two groups based upon their vector relationships as well as symptomatology. One group would include the crinkle and droop types of viruses which are relatively non-persistent in their insect vectors, being retained by them for less than 24 hours. In the second group would fall the relatively persistent viruses which are retained by their vectors for 24 hours or longer and which would include at least one yellows virus. Work is in progress toward the isolation of discrete viruses and a study of their vector relationships.

The results here described are essentially similar to those that have been reported from England (3, 4). The question of whether any of the viruses that have been isolated in England or elsewhere are identical with any present in California must await a direct comparison of the vector-virus relationships and symptomatology on common indicator plants.

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DIVISION OF ENTOMOLOGY, CALIFORNIA AGRICULTURAL EXPERIMENT STATION, BERKELEY

WILD STRAWBERRIES AS A SOURCE OF STRAWBERRY VIRUS INFECTION[1]

P. W. Miller

Certain wild strawberry species are very susceptible to strawberry virus diseases. In 1942, Harris and King (1) reported that a strain of Fragaria vesca from England was very sensitive to certain strawberry viruses. A strain of F. virginiana has also been reported by Hildebrand (2) to be quite susceptible to some strawberry viruses. In 1949, Thomas (3) reported that a strain of F. vesca from California, called var. californica, was likewise quite sensitive to certain strawberry virus diseases.

While there is no question of the virus sensitivity of these strains of wild strawberries, no one has as yet determined to what extent the various species growing in the wild carry the viruses. This is important as they may be sources or reservoirs of viruses that cause primary infection of cultivated strawberries and in that case control practices may have to be modified.

Table 1. Extent and distribution of virus in wild strawberries in Oregon, 1950.

Species and location	Plants Tested	Plants Infected	Species and location	Plants Tested	Plants Infected
	Number	Percent		Number	Percent
F. chiloensis:			F. ovalis (Cont.)		
Near Seaside	9	11.1	Near Nashville	4	25
Near Gleneden Beach	6	0	Near Toledo	1	0
Near Depoe Bay	4	25	Near Eddyville	4	0
Near Agate Beach	4	0	Near Burnt Woods	4	0
Near Newport	7	0	Near Blodgett	8	0
Near South Beach	7	0	Near Wren	9	0
Near Seal Rocks	9	0	5 miles N. of Corvallis	2	0
Near Waldport	10	0	5 miles W. of Corvallis	4	0
Near Yachats	13	15.4	College Farm, Corvallis	14	7.1
4 miles N. of Florence	16	12.5	Near Tangent	3	0
Near Charleston	2	0	Near Lacomb	4	0
Near Bandon	3	25	Near Alsea	4	0
Near Toledo	1	0	Near Seneca	3	0
Near Eddyville	1	0	Near Depoe Bay	1	100
F. ovalis:			F. vesca:		
Near Banks	2	0	Near Banks	4	0
8 miles E. of Gresham	2	0	7 miles N. of North Plains	3	0
Near Beaverton	5	0	Near Dee	6	0
Near Laurel	4	50	Near Gresham	1	0
Near West Linn	2	0	Near Sandy	4	0
3 miles S. of Estacada	5	40	Near Forest Grove	5	0
Near Newberg	4	25	Near Laurel	1	0
6 miles S. E. of Silverton	5	20	5 miles N. of Newberg	4	0
Near Hullt	5	0	Near Scotts Mills	1	0
Silver Creek Falls State Park, near Silverton	4	0	Near Salem	1	0
Near Hazel Green	10	0	10 miles E. of Stayton	4	0
Near Salem	1	0	Near Toledo	1	0
10 miles E. of Stayton	7	0	Near Blodgett	3	0
Near Lyons	4	0	Near Tangent	1	0
Near Logsdon	5	0	5 miles E. of Tidewater	5	0
Near Siletz	5	0	10 miles W. of Alsea	3	0
Near Norton	1	0	Near Blachly	3	0

[1]Cooperative project between the Division of Fruit and Vegetable Crops and Diseases, U. S. Department of Agriculture and the Oregon Agricultural Experiment Station; published with the approval of the Director of the Oregon Agricultural Experiment Station as Technical Paper No. 669. Contribution of the Department of Botany and Plant Pathology.

Experiments were accordingly carried on in 1950 to determine whether healthy looking plants of various wild strawberry species (Fragaria chiloensis, F. ovalis, and F. vesca) native to Oregon were infected with virus, and therefore could serve as sources of virus infection of cultivated varieties. In these investigations, plants from several different clones of wild strawberries were collected from over 50 different locations in Oregon, scattered from Portland to Eugene and from the Pacific Coast to central Oregon. To ascertian whether virus was present in the plants, the majority of them were stolon-grafted to healthy virus-sensitive F. vesca indicator plants. In certain cases, non-viruliferous strawberry aphids were caged on the plants and after they had fed for a while they were transferred to F. vesca indicator plants. The results are given in Table 1.

As shown by the data given in Table 1, viruses were found in plants of F. chiloensis from 5 of 14 locations and in those of F. ovalis from 7 of 31 widely scattered locations in Oregon. While the percentage of infected clones of both these species in any one location was in general relatively low, in one case half of the clones tested were found infected. Repeated tests with these clones, of course, might reveal virus in additional ones. Some of the native plants having virus were collected far from any cultivated strawberries. It seems likely that these two native species are original hosts of at least some of the virus of the strawberry. It is evident also that there is sufficient infection in these two species to constitute a source of reservoir of virus that could cause infection of cultivated strawberries growing nearby. The presence of seemingly virus-free clones raises the question as to whether such clones may be immune or have only escaped infection. In both species the viruses are "latent" or masked and the plants do not show any visible symptoms of disease.

The failure to find viruses in native F. vesca is difficult to understand as some strains of this species growing in Oregon are very susceptible to virus infection. It may possibly be that the infected clones are quickly killed and that those remaining are virus-free, or that the aphid that spreads the virus does not colonize on this species in the wild.

From results of this investigation, it would appear that the presence of wild strawberries in the vicinity of cultivated fields will henceforth have to be taken into account in the general control program.

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U. S. BUREAU PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS, OREGON

STUDIES ON THE DETECTION OF VIRUSES IN THE LEAVES OF STRAWBERRIES BY THE LINDNER STAINING PROCEDURE [1]

P. W. Miller and Franklin D. Aldrich

Recently Lindner and his associates (1, 2) reported the discovery of a simple color test for the detection of virus diseases in certain woody plants. They stated that this color reaction appears to be associated with the accumulation of phenolic compounds in infected tissues. The following virus-infected plants were found to give a positive staining reaction: peaches, cherries apricots, plums, apples, pears, strawberry, roses, and high bush cranberry. Negative results were obtained with all viruliferous herbaceous plants tested except strawberry.

Extensive tests of the applicability of the Lindner staining technique for the detection of viruses in strawberries were conducted by the writers in 1950. More than 500 plants including 3 species of wild strawberries (Fragaria chiloensis, F. vesca, and F. ovalis), 8 varieties of cultivated strawberries (Marshall, Brightmore, Donner, Lassen, Rockhill, Corvallis, Sierra, and Northwest) and over 25 unnamed hybrids were subjected to test.

METHODS

Essentially the procedure described by Lindner, Kirkpatrick, and Weeks (2) with minor modifications was used. In general, the process consists of chlorophyll extraction from whole leaves and treatment of the leaves first with warm 1-normal sodium hydroxide solution and finally with lactic acid. The detailed procedure is as follows: Leaves from plants to be tested are boiled in an extraction solution consisting of 700 ml. of 95 percent alcohol, 20 ml. of commercial formaldehyde, and 230 ml. of distilled water. A 500 ml. Erlenmeyer flask under a reflux condenser was found to be a convenient vessel for treatment. The chlorophyll-extraction time varies from several minutes for young leaves to more than an hour for older leaves of species such as Fragaria chiloensis. After decolorization has occurred, the leaves are transferred to petri dishes and warmed over a steam bath and a small volume of 1-normal sodium hydroxide solution is poured over the leaves. If the leaves are viruliferous, a blue or greenish-blue pigment should develop after a few minutes' treatment. Generally, five minutes in sodium hydroxide suffices for maximum coloration. The leaves are then covered with 85 percent lactic acid, which changes the blue pigment to a more permanent red and renders the leaf tissues more transparent.

RESULTS

The results from more than 500 tests show that leaves from plants with severe symptoms of yellows and severe symptoms of crinkle stain positively when subjected to the Lindner staining test. The conducting tissues of leaves severely affected with yellows stain deep pink or red, the vasculars in the midribs and primary veins showing the most intense coloration (Figure 1, A). In leaves of plants severely affected with crinkle, on the other hand, localized or diffused patches of red or pink occur in the lamina with very little if any staining of the veins (Figure 1, B).

There is considerable difference in the intensity of the stain in leaves of varying ages, the older infected leaves staining more intensely than the younger ones. This would indicate a gradual build-up with age of the leaves of polyphenols or of other compounds which stain.

However, while definite positive stain reactions were always obtained in known cases of severe infection with yellows or crinkle, conflicting color reactions were obtained on leaves from plants mildly affected with yellows and with crinkle. Thus, on one occasion the leaves of a plant mildly affected with yellows stained positively. In a subsequent retest of leaves from the same plant, made several weeks later, a negative reaction was obtained.

It is recognized that the difference in stain reaction may possibly be associated with fluctuations in the virus concentration within the tissues from time to time. This is known to take place in certain other plants infected with viruses. Even so, the variation in the stain reactions of a given plant from time to time constitutes a limitation in the value of the test.

Moreover, a red pigmentation in the leaf serrations and a light pink coloration of the lamina were frequently obtained in the leaves of seedlings of Marshall strawberry indicated to be virus-

[1] Published as Technical Paper No. 668 with the approval of the Director of the Oregon Agricultural Experiment Station. Contribution of the Department of Botany and Plant Pathology.

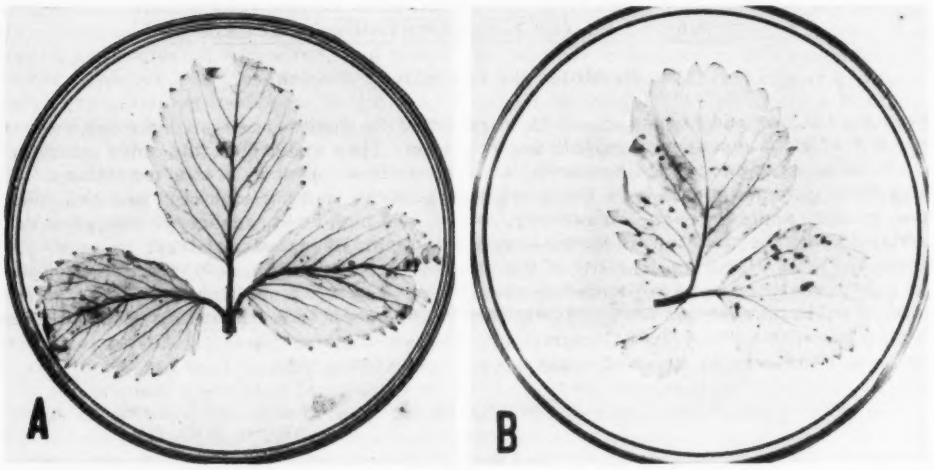

FIGURE 1. Viruliferous Marshall strawberry leaves cleared and stained by Lindner staining procedure: A. Leaf severely affected with yellows; B. Leaf severely affected with crinkle.

free by a negative reaction when runner-grafted to virus-sensitive healthy Fragaria vesca plants (Figure 2). Lindner (2) reported that the red pigmentation in the leaf serrations is a glandular reaction which should be disregarded. Be that as it may, the fact that such areas in apparently healthy plants stain positively is a complicating factor which cannot but detract from the usefulness of the test.

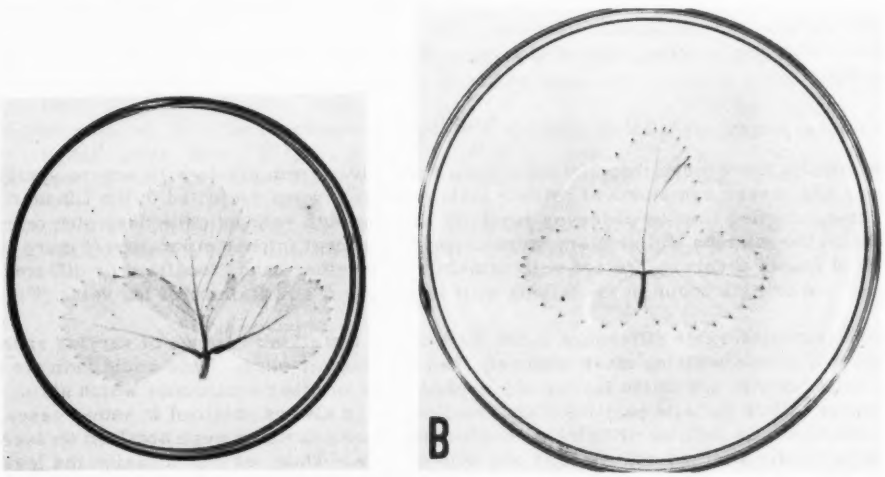

FIGURE 2. A, B, Leaves from healthy strawberries cleared and stained by Lindner staining procedure. Note that in B the serration tips are stained (red).

Leaves of plants affected with powdery mildew also gave a mild staining reaction, light pink patches occurring in leaves where mildew lesions were located.

The possibility of mechanical injuries being involved in the formation of substances which give a staining reaction was also investigated. Several methods of injuring healthy leaves including abrasion and perforation were tried. No color was produced about any of these wounds, even after appreciable time had elapsed before testing.

From the foregoing it is concluded that the Lindner staining procedure as herein described

has definite limitations with respect to the detection of strawberry virus diseases. Plants showing severe symptoms of crinkle or of yellows can be positively identified by this technique but plants mildly affected with crinkle or yellows cannot be reliably detected. The Lindner staining procedure may possibly be of some value as a fast method of separating severely infected plants from less severely infected ones, and it may also have value in diagnosing the relative amounts of virus infection in a given lot of plants. However, it does not appear to be a reliable method for separating healthy from infected plants.

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BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, UNITED STATES DEPARTMENT OF AGRICULTURE, OREGON STATE COLLEGE, CORVALLIS, OREGON

FURTHER STUDIES ON THE RESPONSE OF STRAWBERRY VARIETIES AND SELECTIONS TO STRAINS OF THE RED STELE ROOT DISEASE FUNGUS

D. H. Scott, W. F. Jeffers, George M. Darrow, and D. P. Ink

Relatively few commercial varieties of strawberries are resistant to the red stele root disease. All that are resistant except Aberdeen have been obtained through breeding work and have Aberdeen as the source of resistance.

In a previous report (2) it was shown that there are two known strains the red stele fungus (Phytophthora fragariae Hickman), as indicated by the response of certain strawberry varieties and selections when tested in different soils infested with that fungus. Aberdeen, Fairland, Pathfinder, Sparkle, and Temple were resistant to one strain (strain A) but were susceptible to the other strain (strain S). Md. 683 and Md. 1972 were susceptible to strain A, but resistant to strain S. None of the varieties tested were resistant to both strains.

The studies reported here were conducted in the winter of 1949-50 and dealt with some varieties not listed above and with ten additional selections.

Materials and Methods

Inoculation tests were conducted in the same manner as in the earlier study (2). Two greenhouse benches were filled with soils infested with the red stele fungus: one with strain A and the other with strain S. Previous experience with these same soils had shown them to be thoroughly infested with the disease organism. Single 10-plant plots were used in each bench for most varieties and for part of the selections. Suwannee was planted in duplicate plots in each bench as the check variety, as it was known to be highly susceptible to both strains of the fungus.

A rating system ranging from 1 to 10 for degrees of infection was used to classify the plants. (1). In class 1 the entire root system was completely killed. Plants in class 5 had all roots invaded, but few of the steles were infected the entire length of the root. Class 10 consisted of

Table 1. Reaction of strawberry varieties and selections to strains A and S of the red stele fungus in greenhouse benches.

Variety or selection [a]	Number of plants set	Strain A		Strain S	
		Number of plants examined [b]	Rating	Number of plants examined [b]	Rating
Climax	20	9	8	9	1
Fairland	20	9	8	10	1
Marshall	20	10	5	8	2
Redcrop	20	10	8	7	3
Suwannee	40	20	1	19	2
Vermilion	20	10	10	6	1
Md. 1972	20	10	3	10	10
US 3512	20	9	8	6	2
US 3531	20	9	3	9	9
US 3535	20	10	7	10	6
US 3538	20	10	7	10	8
US 3558	20	10	1	10	1
US 3559	20	10	3	10	5
US 3628	10	5	4	4	4
US 3631	10	5	5	4	6
US 3636	10	5	3	5	7
US 4243	10	5	2	5	8

[a] Parentage of selections: Md. 1972 = Blakemore x Md. 683 (Scotland BK46 x Fairfax); US 3512, US 3531, US 3535, US 3538, and US 4343 = Fairland x Md. 683; US 3558 = US 3374 (Dorsett x Scotland CC18) x Oreg. 1775 (Oreg. 1188 (Clark x Fairfax)) x Oreg. 1088 (Blakemore x Oreg. 186 (Marshall x Fragaria chiloensis)); US 3559 = US 3374 x Oreg. 1491 (767 (Corvallis x US 836) x Narcissa); US 3628, US 3631, US 3636 = Temple x US 3374.

[b] A few plants died from undetermined causes prior to examination.

plants having no visible evidence of infection. Plants in classes 6 to 10 were considered to range from resistant to highly resistant. Fairland and Redcrop, both of which rated 8 in resistance to strain A, for example, are seldom infected under field conditions. All ratings were made by one person.

Results

The results are given in Table 1. Suwannee, as previously found, was susceptible to both strains. Climax, Fairland, Redcrop, and Vermilion were all very resistant to strain A and susceptible to strain S. Vermilion seemed to be somewhat more resistant to strain A than any of the other varieties under test. It is of interest to note that Marshall was not so susceptible to strain A as was Suwannee.

The selections used were from the breeding work for red stele resistance and were saved as resistant seedlings from field or greenhouse bench tests involving strain S of the organism (3). Md. 1972 was susceptible to strain A, but showed no infection with strain S. Most of the other selections are from progenies in which one parent was resistant to strain A and the other to strain S, such as Fairland x Md. 683. These selections had not had a previous test, however, for resistance to strain A.

The reaction of the selections to the two strains is of interest in three respects: Three of the selections had a resistance rating of 7 or 8 to strain A; two of them proved to be as susceptible as Suwannee to strain S; and finally, and most important, selections US 3535 and US 3538 were moderately resistant to both strains of the organism. This indicates that it should not be difficult to originate strawberry varieties having resistance to both known strains of the red stele root disease fungus.

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DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, BELTSVILLE, MARYLAND, AND MARYLAND AGRICULTURAL EXPERIMENT STATION, COLLEGE PARK, MARYLAND

RELATIVE RESISTANCE OF SOME BLUEBERRY VARIETIES AND SELECTIONS TO STEM CANKER IN NORTH CAROLINA

J. B. Demaree and E. B. Morrow

Stem canker in blueberries caused by the fungus Physalospora corticis Demaree & Wilcox is southern in its adaptation and distribution. Most cultivated fields of the southeastern highbush blueberry (Vaccinium australe Small) in North Carolina and most plantings of unselected seedlings of the rabbiteye species (V. ashei Reade) in Alabama, Mississippi, Florida, and Georgia are more or less infected. The disease has also been found in wild bushes in places both remote from and near cultivated plantings, indicating that the causative organism is indigenous to these southern sections. It has not been reported from any northern blueberry-growing State.

The stem canker fungus causes considerable damage to the bushes of some varieties by girdling and killing canes and scaffold branches, thus materially reducing the fruiting potentiality of the plants. This damage is sufficiently great in some otherwise desirable varieties to make them unprofitable to grow. The loss is probably not so great now as formerly, for in many established fields the extremely susceptible varieties have been taken out and replaced with more resistant ones.

The early planted fields of blueberries in North Carolina, planted some 25 or 30 years ago, were set with plants propagated in New Jersey. Even now some growers import New Jersey-grown plants for setting new fields. Since stem canker has never been seen in New Jersey, we may assume that plants grown there were free of the disease when sent to North Carolina. Circumstantial evidence indicates that at least the early planted fields in North Carolina were infected by inoculum spread from diseased wild bushes growing near by. It is not known how soon those early fields became infected, as the disease was unrecognized prior to 1938. At that time, the disease had become well established in a few of the oldest plantings.

Extensive spread may have resulted from propagating new plants from cuttings from infected fields. Commercial propagation is accomplished by using hardwood cuttings of the previous season's growth. Infected cuttings produce infected plants. The causative fungus may even spread to other plants in the propagating frame and later in the nursery. Blueberry nurseries are usually located within or adjacent to old plantings, furnishing another chance for infection of plants before permanent setting. Consequently, it is not unusual to find all plants of susceptible varieties infected to the point of sustaining injury by the time they reach bearing age.

Fortunately, very little propagating material originating in the South was sent North; and the disease so far as is known has been confined to the southern areas. However, there is no known reason why the stem canker fungus will not thrive and spread in more northern regions if once introduced and established.

Perithecia and pycnidia are produced in abundance on cankers of all ages. Spores are expelled from the asci when they are wet and it is thought that rain-spatters disperse the spores through short distances and that drifting rain and wind carry them greater distances.

New infections of the canker fungus appear on the current season shoots as reddish, broadly conical swellings that do not enlarge much during the first season; thereafter their boundaries gradually extend year after year as the mycelium invades new tissues.

Varieties of blueberries exhibit a wide difference in resistance to the blueberry stem canker fungus. A few take the disease so readily and sustain such extreme injury that they cannot be grown profitably in the South. A few other varieties are highly resistant and thrive in fields where susceptible varieties fail.

Symptoms of stem canker (Fig. 1) vary considerably on different varieties. On very susceptible varieties like Cabot and Pioneer the fungus penetrates the cortex, cambium, and finally the wood and produces extensive swollen cankers with deep fissures and cracks frequently involving the entire circumference of the stems (Fig. 1, A). In less susceptible varieties, such as Rancocas, the cankers may be extensive and the surface roughened and cracked, but they are not conspicuously swollen. On more resistant varieties, the stem cankers are scattered and slowly increase in size or they may be entirely absent on the stems and only a few small ones may occur on the branches. On very resistant varieties, the fungus may become established in small shoots but die as the tissues age about the second year.

The varieties used during the early history of the blueberry industry in New Jersey were propagated by Miss Elizabeth White, Whitesbog, New Jersey. Individual wild plants selected on account of high production and the size, color, and dessert quality of their fruit were named. The varieties named later resulted from crosses made by the late F. V. Coville, of the U. S. Department of Agriculture. Among the varieties propagated from the wild are Adams, Chatsworth,

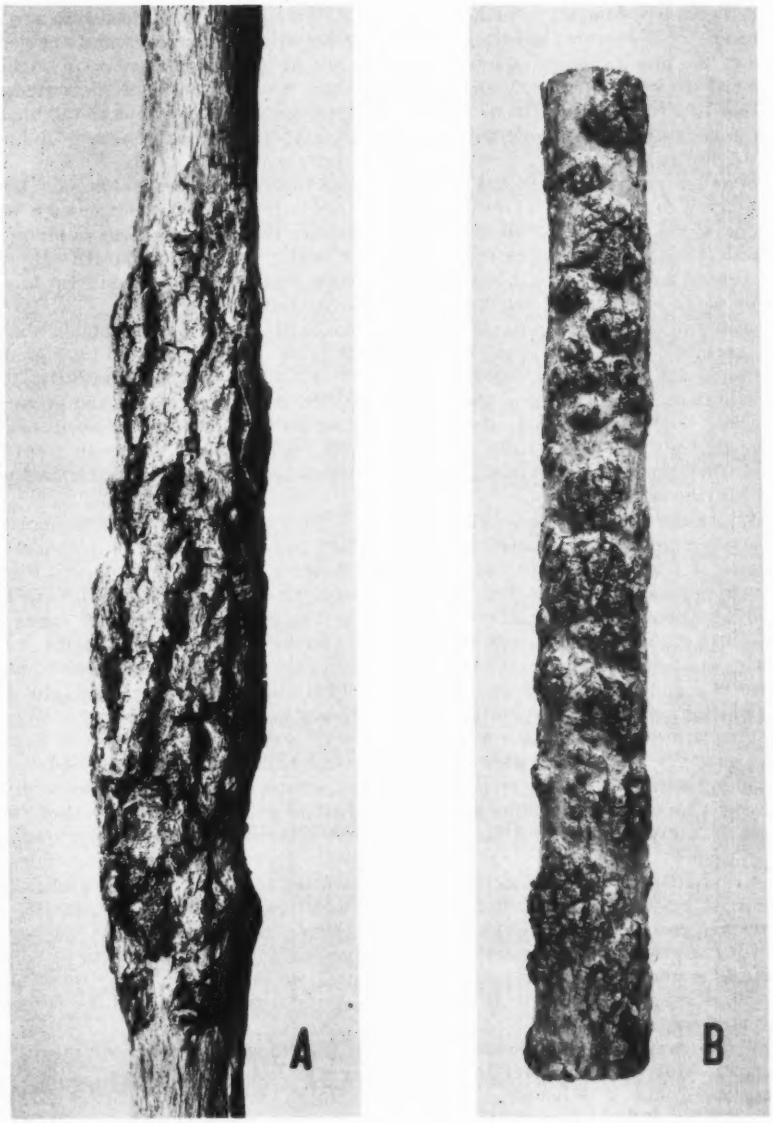

FIGURE 1. Two common types of stem canker. A, continuous and swollen with long deep fissures involving the entire circumference of the stem. B, small separate cankers usually showing first on the south and west sides of the stem and eventually forming a continuous canker.

Dunfee, Grover, Harding, Rubel, and Sam. Of these, Rubel has proved to be highly desirable as a parent of later and better varieties. Rubel entered into the makeup of Atlantic, Berkeley, Burlington, Concord, Coville, Dixi, Jersey, June, Murphy, Pemberton, Rancocas, Scammell, Stanley, Wolcott, and Wareham.

The older rabbiteye blueberry plantings in west Florida and south Alabama are mostly not of clonal composition but are of seedlings dug from the wild along rivers and creeks in those areas. Some of the plantings are now 40 years old and have a great number of canker-infected bushes. Many of the bushes are extremely sucseptible, with solid cankers covering the stem from the ground line to a height of 4 or 5 feet. Approximately 25 percent of the bushes in some plantings are canker-free after constant exposure to infection for many years, indicating that they must be highly resistant.

About a dozen varieties of selected wild bushes of the rabbiteye species have been named. Even though those who made the selections were unaware that the abnormal rough bark on blueberry canes was a symptom of an infectious disease, they nevertheless selected for propagation only those wild bushes that were canker-free or nearly so. Of the named rabbiteye varieties now propagated and planted, all are either immune from or highly resistant to canker. Seven varieties were surveyed in North Carolina during this study.

Only a limited number of selections have been made from the wild highbush blueberry (Vaccinium australe) in southern areas in an attempt to obtain material with a high degree of disease resistance and good berry qualities. Mr. Beck Crabbe, working with Dr. Coville, made several collections in North Carolina about 1927. These were propagated and grown under selection numbers such as Crabbe 1, Crabbe 2, and so forth. Dr. Coville used these selections in a number of crosses and planted the seedlings in North Carolina. Although stem canker was not recognized when the native North Carolina selections were made, they transmitted canker resistance in varying degrees.

From Dr. Coville's crosses two new varieties -- Murphy (NC-255) and Wolcott (NC-262) -- have recently been named and released for propagation and trial. Parents of these varieties include Crabbe 4, a highly resistant North Carolina selection from the wild, and Weymouth and Stanley, two very susceptible varieties. Both new varieties are highly resistant to stem canker; and if they prove to be of commercial value otherwise, as indicated during 10 years of testing, they should rapidly replace the present early-fruiting varieties in North Carolina.

The relative susceptibility of most blueberry varieties and unnamed clones to stem canker has been known in a general way for several years. This information was accumulated as the result of observations made by several persons in widely separated blueberry plantings. Usually no more than one-half dozen varieties were available for examination in any one field, although there has been need for more accurate information concerning all varieties grown. An ideal location for making a survey of this type would be one where many varieties were grown within a restricted area where the topography and soil conditions were similar and other factors favoring infection were fairly constant. A field located on the farm of Mr. Gale Harrison, Ivanhoe, North Carolina, and established several years ago by the North Carolina Agricultural Experiment Station and the U. S. Department of Agriculture for testing blueberry seedlings has fulfilled these requirements better than any other known. In addition, a field near Magnolia, North Carolina, contained plants of 8 varieties and 40 selections, and another near Atkinson, North Carolina, with 2 varieties and 23 selections, were available for rating. A survey of these fields in January 1949 is probably the most complete one made so far. Each entity was classified on the basis of ratings ranging from 1 to 10 and the value recorded was that of the combined judgment of two observers.

Of the highbush varieties available for rating, the following are grown commercially in North Carolina: Wolcott, Murphy, Scammell, Atlantic, Jersey, Rubel, Rancocas, Dixi, June, Pemberton, Burlington, Concord, Pioneer, Stanley, Weymouth, and Cabot. If it is correct to assume that the rating of 7 represents the minimum resistance for commercial tolerance, then it may not be advisable to plant any of the highbush varieties in the South except Wolcott, Murphy, Scammell, Atlantic, and Jersey. The Rancocas, a variety widely grown in North Carolina, is on the border-line for stem canker resistance, but it is productive, dependable, and highly resistant to the destructive virus disease known as stunt and will not be discarded on account of its susceptibility to canker alone.

All seven varieties of the rabbiteye blueberry listed in Table 1 are suitable for growing in North Carolina from the standpoint of stem canker resistance.

Table 2 is of interest because of the horticultural importance of the blueberry clones listed. Since the canker survey was made in 1949, clones DN-76 and U-85 have been named and released for unrestricted propagation. DN-76 is now the variety Coville and U-85 is Berkeley. BL-32 may be named later and F-72 and BM-22, which possess outstanding horticultural characteristics,

Table 1. Stem canker resistance ratings of blueberry varieties, Gale Harrison field, Ivanhoe, N. C., 1949.

Variety	Resistance rating*	Number plants examined	Parentage
Highbush varieties:			
Adams	10	5	Wild New Jersey seedling
Harding	10	5	" " " "
Wolcott	10	2	Weymouth x F-6 (Stanley x Crabbe 4)
Crabbe 6	8	?	Wild North Carolina seedling
Murphy	8	5	Weymouth x F-6 (Stanley x Crabbe 4)
Scammell	8	2	(Brooks[a] x Chatsworth[b]) x Rubel
Sam	8	5	Wild New Jersey seedling
Atlantic	7	10	Jersey x Pioneer
Jersey	7	5	Rubel x Grover
Taylor	7	3	Wild Michigan seedling
Rubel	6	5	Wild New Jersey seedling
Wareham	6	5	Rubel x Harding
Rancocas	6	5	(Brooks x Russell[c]) x Rubel
Dixi	5	5	(Jersey x Pioneer) x Stanley
Katharine	5	5	Brooks x Sooy[d]
June	5	13	(Brooks x Russell) x Rubel
Pemberton	5	9	Katharine x Rubel
Burlington	4	5	Rubel x Pioneer
Concord	4	6	Brooks x Rubel
Grover	4	5	Wild New Jersey seedling
Piooner	4	5	Brooks x Sooy
Stanley	4	3	Katharine x Rubel
Weymouth	4	6	June x Cabot
Cabot	2	5	Brooks x Chatsworth
Rabbiteye varieties:			
Black Giant	10	10	Wild seedling from Florida
Clara	10	1	" " " Georgia
Hagood	10	10	" " " Florida
Mineola	10	10	" " " Georgia
Myers	10	10	" " " Georgia
Sapp Early	10	10	" " " Florida
Suwannee	9	10	" " " Georgia

* Definition of resistance ratings:

1. Numerous cankers or entire involvement of base of old canes, accompanied by deep-fissured swellings and some cane necrosis.
2, 3, 4, and 5. Abundant cankers but progressively less in number, but sufficient to shorten life of bushes. All below commercial tolerance.
6. Cankers mostly of shallow type, including the "blister" type, and confined mostly to smaller shoots. Commercial tolerance doubtful.
7. Few scattered cankers, of the shallow type, on small shoots. Base of canes free of canker. Injury slight. Life of bush not shortened.
8. A very few small shallow active cankers on twigs.
9. One or two small, but inactive, cankers.
10. No cankers.

[a]The Brooks was a wild New Hampshire highbush
[b]The Chatsworth was a wild New Jersey highbush
[c]The Russell was a wild New Hampshire lowbush
[d]The Sooy was a wild New Jersey highbush

Table 2. Stem canker resistance ratings of highbush blueberry selections made in New Jersey. Gale Harrison field, Ivanhoe, N. C., 1949.

Selection Number	Resistance rating [a]	Number plants examined	Parentage
BL-32	7	15	Z-13 (Rancocas x Carter [b]) x Stanley
F-72	7	13	Wareham x Pioneer
BG-39	6	1	GM-37 (Jersey x Pioneer) x Stanley
AM-54	5	3	GM-37 (Jersey x Pioneer) x Stanley
AR-64	5	3	GM-37 (" x ") x "
AR-82	5	20	Concord x 1239-E (Harding x Rubel ?)
AW-35	5	1	GM-37 (Jersey x Pioneer) x Concord
DN-76	5	6	GM-37 (" x ") x Stanley
DQ-53	5	25	GM-37 (" x ") x "
L-25	5	2	GS-149 (" x ") x "
T-72	5	2	Stanley x GS-149 (Jersey x Pioneer)
U-75	5	6	" x " (" x ")
U-85	5	2	" x " (" x ")
V-25	5	4	" x " (" x ")
A-91	4	25	Wareham x Pioneer
BM-22	4	1	Z-13 (Rancocas x Carter) x Stanley
GS-149	4	22	Jersey x Pioneer
O-29	4	10	Scammell x Crabbe 17
V-20	4	1	Stanley x GS-149 (Jersey x Pioneer)
X-58	4	1	" x " (" x ")
R-86	3	4	" x " (" x ")

[a] See Table 1 for explanation of resistance ratings.
[b] Carter parentage unknown.

may also be named. Other clones such as X-58, R-86, and V-25 are probably superior to some varieties now widely grown in northern sections where canker is unknown at present.

These clones were originated by Dr. Coville several years ago by using mostly New Jersey non-canker-resistant varieties and selections as parents. Although the number of selections available for canker resistance rating was small, those available suggest that very little progress could be made in breeding in an attempt to obtain canker resistance by using such as parents. On account of the absence of high canker resistance, none, with the possible exception of BL-32 and F-72, would be suitable for planting in southern sections although some may prove satisfactory if grown in New Jersey, New England, New York, and Michigan.

Breeding for stem canker resistance in North Carolina has centered around four wild southern selections of the highbush blueberry, known as Crabbe 4, Crabbe 6, Crabbe 11, and Crabbe 17. Unfortunately in 1949, none of these were available for canker resistance rating except Crabbe 6, which was given the rating 8. Four or five years earlier Crabbe 4 and F-6 (Stanley x Crabbe 4) had been found to be canker-free. There are no records concerning the canker resistance of Crabbe 11 and Crabbe 17. It may be assumed, however, that they were highly resistant as canker-infected wild bushes of the highbush species in North Carolina are rarely encountered.

Ten separate crosses were made by the late F. V. Coville, using the Crabbe selections as one parent and New Jersey varieties or selections as the other parent; these crosses resulted in many seedlings which were given preliminary testing and 100 were selected for more extensive testing. These last have been given selection numbers ranging from NC-204 to NC-414. All were examined and rated for stem canker resistance in one or more locations in 1949 and their performances are indicated in Table 3. Although the number of selections was small, they may be of some value to those interested in breeding for canker resistance. The Crabbe selection crosses transmitted a high percentage of resistance to the progenies even though the New Jersey variety Stanley, a highly susceptible variety, was a parent or grandparent in eight of the ten crosses. Concord and Weymouth, both also highly susceptible, entered into the other two crosses.

Crabbe 4 transmitted a higher degree of resistance to stem canker than any of the others. If it can be assumed that plants rating 7 to 10 are commercially acceptable, 73 percent of the Crabbe 4 selections, 32 percent of the Crabbe 6 selections, 48 percent of the Crabbe 11 selections,

Table 3. Southern blueberry selections compared with northern varieties and selections, by stem-canker-resistance classes.

Origin of varieties and selections	Number of crosses	Number of varieties or selections	Percent in each class[a]				
			land 2	3 and 4	5 and 6	7 and 8	9 and 10
Southern:							
Crabbe 4 crosses	2	33		6	21	18	55
Crabbe 6 crosses	4	31	20	26	22	10	22
Crabbe 11 cross	1	21	9	24	19	19	29
Crabbe 17 crosses	3	15	27	20	20	13	20
Total	10	100	12	18	21	15	34
Northern:							
Varieties	-	23	4	35	30	22	9
Selections	-	18	0	44	44	12	0
Total		41	2	39	37	17	5

[a]See Table 1 for explanation of disease ratings.

and 33 percent of the Crabbe 17 selections are suitable from the standpoint of canker resistance for growing in the South. If all of the selections containing germ plasm from native eastern North Carolina are combined, 49 percent come within the 7 to 10 resistance ratings. This compares with 22 percent for all northern varieties and selections rated in 1949.

There are several other North Carolina selections possessing excellent fruit characters combined with high stem canker resistance available for unrestricted propagation. There are also 34 selections possessing high canker resistance rated 9 and 10 and 15 other selections rated 7 and 8 from which future southern blueberry varieties may be taken.

Summary

The stem canker caused by Physalospora corticis is second in importance to stunt, a virus disease, as a limiting factor to cultivated blueberry growing in southern regions. The canker is found in nearly all North Carolina fields and renders the most susceptible varieties worthless. Varieties and unnamed clones vary greatly in susceptibility.

Of the several northern blueberry varieties widely grown in the South, none except Atlantic, Jersey, and Scammell are highly resistant to stem canker. The varieties Pemberton, Rancocas, and Rubel are moderately resistant. Cabot, Concord, Dixi, June, Pioneer, Stanley, and Weymouth are highly susceptible and on this account are unsuitable for growing in southern sections.

There is very little foundation material in northern varieties and selections for use by breeders for creating stem canker-resistant sorts. A few selected wild North Carolina highbush blueberry clones, known as Crabbe selections, have proved to be exceedingly valuable for transmitting canker resistance when crossed with northern varieties possessing desirable fruit qualities, even though the northern varieties used are highly susceptible to canker. Two varieties, Murphy and Wolcott, possessing adequate canker resistance, developed from the above parent combination have recently been named and released for unrestricted propagation. A considerable number of unnamed selections of similar parentage, showing high resistance to canker, are now under preliminary testing; and some have sufficient horticultural merits to be named and distributed for commercial use.

There is no immediate problem concerning stem canker resistance in the rabbiteye blueberry as all standard varieties are either immune or highly resistant.

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GRAPE DEAD-ARM CONTROL

Wm. B. Hewitt

Dead-arm disease of grapevines has required some form of control, particularly in the Sacramento and Lodi areas of California. The severity of the disease has varied with the season since it was first reported in 1935 (1). Table grape varieties of Vitis vinifera, namely Tokay, Olivette blanche, and Cornichon, have been most severely affected by the disease in these areas.

Leaf, shoot, cluster, and cane spotting similar to that shown in Figure 1 has been the most conspicuous symptom of the disease. Killing of spurs, arms, and even entire vines has occurred after two or more consecutive seasons very favorable to the development of spring infections.

FIGURE 1. A shoot of Olivette blanche grapevine which shows leaf, stem and form spotting caused by the dead-arm fungus Cryptosporella viticola Shear.

Experimental control plots have been maintained for the past several years to compare various pruning techniques and chemical sprays. A sodium arsenite spray applied in the late dormant season has been found to be the most effective and consistent control of spring infections (2). The sodium arsenite solution used was a commercial compound commonly sold as a weed-killer or for spraying grape vines to control black measles. The compound contained an equivalent of four pounds of arsenic trioxide per gallon. For dead-arm control, it was used at the rate of three quarts in 100 gallons of water. The dangers of the spray should be pointed out because it is poisonous and caustic. In some seasons, it has caused considerable damage to buds, spurs, and canes when the sodium arsenite solution apparently penetrated pruning wounds and leaf scars that had not plugged or healed before spraying (3). Spray damage has been reduced by applying the spray as late in the season as possible. If the spray was applied before pruning, the vines were pruned several days after spraying, preferably even after a rain; and if the spray was applied after pruning, then the vines were pruned early in the fall and the spray application was made as late in the dormant season as possible or just before the buds began to open in the spring.

During the spring of 1950, dinitro sec. butylphenol triethanolamine 38% (DN-289) was compared with sodium arsenite, sodium dinitro-ortho-cresolate 32% (Elgetol), and ferric dimethyldithiocarbamate (ferbam) 76% (Fermate). Concentrations of materials per 100 gallons of water were as follows: DN-289, 2 quarts; sodium arsenite, 3 quarts, or an equivalent of 3 pounds of arsenic trioxide; Elgetol, 1 gallon; and Fermate, 3 pounds. Treatments were

applied to individual rows of 20 to 30 vines, randomized, and each separated by a row of non-sprayed control vines. They were replicated in three separate blocks. Results were obtained by counting the total number of shoots, and the number of diseased shoots on each of ten separate vines in each row. The vines averaged from 37 to 40 shoots each. Non-sprayed control vines averaged 13.7 diseased shoots per vine; sodium arsenite, 1.4; Elgetol, 5.7; Fermate, 8.7; and DN-289, only 2.6.

The severity of the disease in 1950 was only moderate, but it was fairly evenly distributed over the plot and the disease counts were uniform and consistent. DN-289 appears to have considerable promise as a spray control for the spring infections of dead-arm and is also probably less hazardous than sodium arsenite.

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THE CONSTRICTION DISEASE OF PEACH IN MARYLAND

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An outbreak of the constriction disease of peach occurred on the Eastern Shore of Maryland in 1949. A report by Roberts (2) in 1940 indicated that this disease had caused serious injury in Delaware and had been a problem for a number of years. Roberts (3) considered the pathogen to be Diaporthe eres Nitsch. with an imperfect stage, Phomopsis. In a review of literature Hildebrand (1) under the heading "Fusicoccum canker" considered the disease a minor one in New York.

FIGURE 1. Constriction cankers on 1- and 2-year old peach wood. (Collected August 1950).

In Maryland, the disease was noticed by a peach grower near Westover in the Fall of 1949. The variety Golden Jubilee was severely infected while Elberta and Hale Haven were comparatively disease-free. Later in the Spring of 1950, other orchards were found with constriction canker in the Golden Jubilee variety. From samples, J. A. Stevenson of the Division of Mycology and Disease Survey confirmed the fungus to be identical to the Phomopsis studied by Roberts.

Cankers developed at the lateral buds on the succulent (1949) growth. On Golden Jubilee, many of the cankers girdle the twig causing wilting and subsequent death. The disease was found on bearing vigorous trees and is not confined to old weakened trees.

Growers and a number of investigators (unpublished results) have not succeeded in the control of this disease either by spraying or by pruning. During 1950, growers removed the Golden Jubilee variety from their orchards. Cankers on resistant varieties are not so prominent and a callus forms. In certain seasons favorable for this disease constriction canker is of major importance on the Eastern Shore. Growers should avoid susceptible varieties when planting new orchards.

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NUT DISEASES IN OREGON IN 1950

Paul W. Miller

WALNUT DISEASES

Walnut Blight (Xanthomonas juglandis (Pierce) Dowson). Walnut blight was present in only very limited amounts in 1950 in most localities in Oregon. Losses from this disease in non-sprayed orchards varied according to the locality from a negligible amount up to 25 percent. It is estimated that not more than 4 percent of the 1950 Oregon walnut crop was destroyed by this disease. This is one of the smallest crop losses from walnut blight in Oregon on record.

Mushroom Root Rot (Armillaria mellea Fr.). This disease caused the death of some seedling Persian walnuts and of a limited number of Franquette walnuts grafted on Hind's black walnut rootstock (Juglans hindsii Jeps.). The finding of mushroom root rot in trees grafted on this rootstock is noteworthy as it is considered to be highly resistant to this disease.

Leaf Scorch (non-parasitic). This leaf disorder was widely distributed in the Pacific Northwest in 1950, occurring to a greater or less extent in many Persian walnut orchards in Oregon. In orchards on soil types very deficient in boron it was exceedingly prevalent, causing a marked decrease in the functional leaf area through death of the tissues and premature defoliation. Soil applications of borax over a period of years and in sufficient amounts generally decrease the amount and severity of this disorder.

Blackline (Girdle) of Grafted Walnuts (non-parasitic). This disorder was responsible for the death of more grafted Franquette walnut trees in 1950 in Oregon than any other disease. The importance of this disorder in the decline of walnuts was indicated by the results of a detailed survey made in 1950 in ten representative, widely scattered, declining grafted Franquette walnut orchards. In these ten orchards, there were found 518 declining trees, 80 percent of which were declining from the effects of the blackline disorder. This trouble was widely distributed, occurring to a greater or less extent in practically all walnut districts in Oregon. All trees found affected were of the Franquette variety grafted on Hind's black walnut rootstock or on hybrids of other species with this one.

Crown Rot (cause undetermined). A crown rot of undetermined cause was found affecting a considerable number of Persian walnuts grafted on Hind's black walnut rootstock in western Oregon. Most of the affected trees were located in hill orchards on "heavy" soil types.

FILBERT DISEASES

Bacterial blight (Xanthomonas corlyina Miller et al.) This disease was widely distributed in 1950, occurring to a greater or less extent in practically all filbert orchards in western Oregon. It was most prevalent in young orchards one to three years of age, where it caused the death of a considerable number of young trees. It also occurred in many older orchards, where it caused buds and twigs to die.

Mildew (Phyllactinia corylea Pers. ex Karst.). More or less of this disease occurred in many orchards in western Oregon in 1950. However, it was of no economic importance as it did not make its appearance until very late in the season. The infected leaves retained their normal green color until the end of the season and persisted on the trees until time of normal leaf fall.

Brown-stain (non-parasitic). This disorder was present in limited amounts in some filbert orchards in western Oregon in 1950. Crop losses from this disorder ranged from less than 1 up to 5 percent, the estimated average loss being about 2 percent.

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RESULTS FROM CO-OPERATIVE SEED TREATMENT EXPERIMENTS ON SMALL GRAINS[1]

J. E. Machacek[2]

In 1950, 40 seed treatments were compared in 59 uniform tests distributed among 15 co-operating stations in Canada and 7 in the United States. Usable data were obtained from 51 of these tests, the balance being affected by adverse weather conditions. The stations and co-operators are listed below.

Canada	United States
Charlottetown, P.E.I. (Dr. G. W. Ayers)	Geneva, N.Y. (Dr. W. Crosier)
Fredericton, N.B. (Mr. J. L. Howatt)	Ames, Iowa (Dr. C. S. Reddy)
Ste. Anne de la Pocatière, Que. (Dr. R. O. Lachance)	Madison, Wisc. (Dr. D. C. Arny)
Winnipeg, Man. (Mr. H. A. H. Wallace)	Brookings, S.D. (Dr. J. T. Slykhuis)
Morden, Man. (Mr. W. J. Breakey)	Fargo, N.D. (Dr. W. E. Brentzel)
Brandon, Man. (Mr. W. H. Johnston)	Bozeman, Mont. (Dr. E. R. Hehn)
Indian Head, Sask. (Mr. J. G. Davidson)	Pullman, Wash. (Dr. C. S. Holton)
Swift Current, Sask. (Mr. D. S. McBean)	
Melfort, Sask. (Mr. H. R. Ballantyne)	
Saskatoon, Sask. (Dr. R. C. Russell)	
Scott, Sask. (Mr. A. G. Kusch)	
Lethbridge, Alta. (Dr. M. N. Grant)	
Lacombe, Alta. (Dr. A. D. McFadden)	
Edmonton, Alta. (Drs. A. W. Henry and L. E. Tyner)	
Beaverlodge, Alta. (Mr. A. A. Guitard)	

Of the seed treatments compared, those now available to the public are named below, and the results from them are shown in Table 1.

1. Aagrano -- Two dust formulations were tested. One, Aagrano 48, contains 0.85% Hg as etoxy-propyl mercury bromide; the other contains 1.7% Hg. Both products are prepared by M. Wiersum, Groningen, Holland. The stronger form is to be marketed in the United States by the Mathieson Chemical Corp., Baltimore, Maryland.

2. Agrox C -- This is a dust containing 2.5% Hg as a mixture of mercury oxide, phenyl mercury acetate, and ethyl mercury chloride. It is made by Plant Protection Ltd., London, England. The Canadian distributors are the Chipman Chemical Co., Winnipeg, Manitoba. The Agrox marketed in the United States contains 6.7% phenyl mercury urea. The distributors are the Chipman Chemical Co., Bound Brook, New Jersey.

3. Ceresan M -- This seed disinfectant was tested both as a dust and a water slurry. It contains 3.2% Hg as ethyl mercury p-toluene sulfonanilide and it is made by E. I. Dupont de Nemours, Wilmington, Delaware. It is distributed in Canada by Canadian Industries Ltd., Montreal, Quebec.

4. Half-ounce Leytosan -- This material was tested as a dust and a water slurry. It contains 7.2% phenyl mercury urea and is made by F. W. Berk & Co., London, England. It is distributed in Canada by Leytosan (Canada) Ltd., Winnipeg, Manitoba.

5. Mercuran -- There are two forms of this product. One of them (Mercuran AS) is applied to seed as a dust; the other (Mercuran AL) is used as a water slurry. Both forms, according to a letter from the Canadian distributors (Charles Albert Smith Ltd., Toronto, Ontario), contain 3.5% Hg, although R. W. Leukel mentions in correspondence that "Information Sheet 505" issued by Delmar Chemicals Ltd., Lachine, Quebec, (the makers of Mercuran) indicates that Mercuran AL contains 7.0% Hg. The results from the co-operative tests referred to in this report suggest that the HG content of the two formulations is about the same.

6. Mergamma C -- This material is reported to contain 2.0% Hg as a mixture of ethyl mercury chloride and phenyl mercury acetate, with 40% gamma benzene hexachloride added to control wireworms. A product sold in the United States under the name "Mergamma" is stated

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[1]Contribution No. 1070 from the Division of Botany and Plant Pathology, Science Service, Department of Agriculture, Canada.

[2]Senior Plant Pathologist.

by the distributors there to contain 2.0% Hg as phenyl mercury urea. The makers and distributors of Mergamma C are the same as those of Agrox C.

7. Panogen -- This product, made and distributed by Panogen, Inc., New York, New York, and Winnipeg, Manitoba, has as its active principle methyl mercury dicyan diamide. Panogen 8 contains the equivalent of 0.8% Hg in a water-miscible fluid base and Panogen 14 1.4% Hg in an oil base.

All of the fungicides tested were applied to contaminated, diseased, or damaged seed of small grains, most of them at several dosages. The seed stocks treated were as follows:

A. Red Bobs wheat artificially contaminated with spores of Tilletia foetida and T. caries (1 part of spores, by weight, to 200 parts of seed).

B. Vanguard oats naturally contaminated by Ustilago avenae and U. kolleri. The natural contamination by smut was supplemented by adding mixed spores of these fungi (1 part spores to 400 parts seed) to the seed.

C. Plush barley naturally contaminated by covered smut (U. hordei) and false loose smut (U. nigra). The seed was mostly of the variety Plush but contained some seed of another variety.

D. Regent wheat in which all the kernels were naturally infected by the seedling blight and root rot fungus, Helminthosporium sativum.

Table 1. Summary of data on the effectiveness of different seed-treatment fungicides in the control of certain seed-borne diseases of wheat, oats, barley, and flax in cooperative experiments at 15 stations in Canada and 7 in the United States in 1950

Treatment	Dosage per bushel				Smut tests (Percentage smutted heads)			Seedling blight tests (Percentage germination)	
	Wheat	Oats	Barley	Flax	Wheat	Oats	Barley	Wheat	Flax
Control (no treatment)	0.00	0.00	0.00	0.00	40.9	17.0	13.6	42.9	34.8
Ceresan M dust	.50	.50	.50	1.50	0.2	0.2	0.4	68.3	42.3
Leytosan dust	.50	.50	.50	1.50	4.1	0.4	0.5	65.6	43.2
Mercuran AS dust	.50	.50	.50	.50	3.9	0.4	0.7	69.0	37.7
" " "	.75	.75	.75	1.00	1.3	0.2	0.4	71.8	43.3
" " "	1.00	1.00	1.00	1.50	0.6	0.1	0.3	74.5	42.8
Aagrano 48 dust	1.00	1.00	1.00	1.00	7.4	0.1	0.9	69.4	37.6
" " "	1.50	1.50	1.50	2.00	1.8	0.1	0.7	72.2	38.6
" " "	2.00	2.00	2.00	3.00	0.5	0.1	0.7	70.5	40.0
Aagrano 250 dust	.25	.25	.25	.50	22.9	0.6	2.5	78.0	39.3
" " "	.50	.50	.50	1.00	6.6	0.1	1.4	69.7	42.1
" " "	.75	.75	.75	1.50	1.3	0.1	0.5	72.3	38.9
Mergamma C dust	1.00	1.00	1.00	1.00	1.9	0.2	0.6	72.0	37.6
" " "	1.50	1.50	1.50	1.50	0.4	0.1	0.5	74.1	42.0
" " "	2.00	2.00	2.00	2.00	0.3	0.1	0.7	66.7	38.4
Agrox C dust	.75	.75	.75	1.50	2.0	0.1	0.3	73.7	40.4
" " "	1.00	1.00	1.00	2.00	1.0	0.1	0.5	72.1	40.1
" " "	1.25	1.25	1.25	2.50	0.4	0.1	0.5	74.0	41.0
Panogen 8 (glycol base)	1.50	1.50	1.50	4.50	0.2	0.1	0.4	71.8	46.5
Panogen 14 (oil base)	.75	.75	.75	1.50	0.4	0.1	0.7	73.6	43.3
Ceresan M slurry	.25	.25	.25	.75	0.2	0.1	0.5	72.2	39.6
" " "	.50	.50	.50	1.50	0.0	0.1	0.5	72.9	44.5
" " "	.75	.75	.75	2.25	0.1	0.1	0.4	70.2	44.1
Leytosan slurry	.25	.25	.25	.75	9.9	6.5	2.9	70.5	37.4
" "	.50	.50	.50	1.50	0.9	4.3	0.8	68.9	37.7
" "	.75	.75	.75	2.25	0.3	2.0	1.5	70.0	37.9
Mecuran AL slurry	.25	.25	.25	.75	15.5	9.4	5.8	65.0	40.0
" " "	.50	.50	.50	1.50	3.7	3.5	1.6	65.1	43.3
" " "	.75	.75	.75	2.25	2.7	2.4	0.7	73.9	45.5
Significant difference 5%					9.7	2.6	3.5	3.2	4.2

E. Royal flax in which about 50 percent of the seeds had been fractured during threshing.

In treating the seed, the dusts were applied on the basis of ounces per bushel, and the fluid treatments, such as Panogen, on the basis of fluid ounces per bushel. The slurries were applied at the rate of 2.3 cc. per pound of seed, the concentrations being equivalent to 0.25, 0.50, and 0.75 ounces of dust to a bushel for cereals, and to 0.75, 1.50 and 2.25 ounces of dust to a bushel for flax.

In Table 1 are summarized the results from 7 tests with covered smut (bunt) of wheat, 12 tests with oat smuts, 14 tests with barley smuts, 9 tests with wheat seedling-blight, and 9 tests with flax seed-rot. As the tests were distributed over a large area and were exposed to a wide variety of soil and weather conditions, the performance of the fungicides tested should be indicative of their performance under a wide variety of farm conditions.

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OAT DISEASES IN ARKANSAS 1949-50

H. R. Rosen

The fall of 1949 was unfavorable for the seeding of winter oats in Arkansas with the exception of the northwest corner, so that for the fifth year in succession the acreage of winter oats was considerably below that of 1944 despite a marked increase in the livestock industry, especially in dairy cows and poultry. The average State rainfall of 8.5 inches for October, when oats are usually sown over most of the State, prevented and delayed seeding to such an extent that in addition to reducing the acreage, stands were poor and very uneven.

Stands were further reduced by excessive rainfall in January and February, particularly in the rice area where the oat acreage is large, and for the first time in many years the oat varietal tests at the Rice Branch Experiment Station had to be abandoned because of the drowning of most of the crop.

Plants which survived and which stood in poorly drained areas showed the following abnormalities (observed during May and June in a statewide survey). There was considerable reduction in stools per plant, reduction in height, often a failure of stools to head, a thickening of stems, reduction in size of blades, and various types of abnormal panicle formation especially supernumerary florets and often sterile spikelets growing out of normally-placed spikelets, partaking of the nature of teratomas. Such plants appeared to be especially susceptible to attacks by the anthracnose organism, which, instead of causing relatively small streaks and blotches on leaves as it usually does, often produced individual lesions that involved most of the sheath and blade.

It was noted by Dr. C. R. Adair and the writer that there appeared to be a difference in varietal reaction to excessive soil moisture. In general the Red Rustproof strains appeared more tolerant or withstood the effect of prolonged "wet feet" better than other types of oats. Thus Ferguson 922, Nortex, and Nortex 107 showed less abnormalities than Fulgrain, Victorgrain, Traveler, Stanton, Letoria, DeSoto, Forkedeer, or Lee, and had better stands.

While this report concerns diseases only, it should be recorded that there were considerable aphid infestation and serious damage by army worms, the latter being especially destructive in the eastern part of the State. There was also a general infestation of leaf hoppers in the fall of the year.

Crown Rust (Puccinia coronata). This disease was far more common in 1950 than it had been during the past few years and doubtless caused considerable reduction in yield especially in the southern half of the State. In the northern half it was held down to a considerable degree on winter oats by dry March weather and by an exceptionally cool March and April. However, on spring oats it was highly destructive and in numerous fields it was estimated that the reduction in yield would run as high as 30 to 40 percent, especially on the Bond derivatives Clinton and Benton.

This is the first year in which considerable rust was found in quite a few fields of Red Rustproofs as well as on Victoria derivatives. Heretofore badly rusted fields of the former type were rarely found; of the latter practically never. In 1950 relatively large fields of Ferguson 922, Delta Red, Nortex 107, and Nortex were observed in various parts of the State in which rust infection ran high as 70 percent of the leaf area when the plants were in the early milk stage. However, the average amount did not run much over 30 percent in severity of infection.

Likewise, on Victoria derivatives such as Traveler, Victorgrain, Fulgrain, DeSoto, and Stanton there were occasional fields found where crown rust ran as high as 30 percent and in the varietal test plots at the Fruit and Truck Branch Station, Hope, the estimated severity went up to 50% of the leaf area (modified Cobb's scale) on DeSoto when the plants were in the milk stage.

While an apparently new race of crown rust was first noted at the University Farm, Fayetteville, in the fall of 1949, producing reactions intermediate on Victoria derivatives and fully susceptible on Bond derivatives (Rosen, H. R. and Murphy, H. C. A race of crown rust apparently new to the United States. Phytopathology 41: 31, 1951), it was surprising to find so much crown rust on Victoria derivatives in various parts of the State in 1950. Evidently this new race or similar races were pretty widely distributed.

Out of 33 collections made in 1950 that have been analyzed for race identification, 19 seemingly involved this new race. Therefore, it appears to have become an immediate threat to a considerable acreage of oats in Arkansas, and probably in at least other southern States where the Red Rustproofs and Victoria and Bond derivatives predominate.

It is also of interest to note that the Victoria derivatives, while showing much more rust than in previous years, were far superior to the Bond derivatives, both of the winter and spring types, in the amount of rust found, and that of the 33 collections thus far analyzed for identifica-

tion, not a single one was found that did not produce a susceptible type reaction on Bond. Fortunately, there are available a number of oat varieties and selections that appear to offer resistance to the new race (see article quoted above) as well as to the older races.

Leaf Spot (Pyrenophora (Helminthosporium) avenae). This disease continues to be one of the most common of all oat diseases, especially on winter oats. While seedling infections are common (PDR 34: 43-44, 1950), they are usually fairly sparse, certainly not nearly so abundant as from heading time to maturity. On the adult plant, any part of the leaf area that is not already occupied by crown rust or anthracnose is almost certain to fall prey to leaf spot. Apparently, whichever organism reaches a susceptible variety first is likely to be the one most prevalent in a particular field. Of course it is not quite so simple, since differences in environmental conditions do have a differential influence on disease prevalence. Does any one know of a variety resistant to leaf spot?

The markedly increased prevalence of leaf spot (as well as of anthracnose) during the past six years or more is probably due to several factors: The much larger State acreage of oats building up the inoculum enormously; the changing to new varieties with probably greater susceptibility; and perhaps most important of all, the comparative rarity of seed treatment with an organic mercurial. With the introduction of Victoria derivatives, which at first were resistant to all the common races of oat smuts, a condition no longer true, growers appear to have become complacent about seed treatment. Losses due to smut are much more discernible to the average grower than those from leaf spot, anthracnose, or even crown rust. As the smut races that attack Victoria derivatives become more prevalent, we may expect greater interest in seed treatment and probably a diminution of leaf spot incidence.

Anthracnose (Colletotrichum graminicola). This disease continues to be common in Arkansas, although it appeared in general to be less abundant in 1950 than in 1949 (PDR 34: 43-44, 1950), probably owing in part at least to the greater abundance of crown rust. It was especially severe in fields that had been flooded, although it was found in every one of the fields in the 58 counties included in the survey, and on almost all plants.

Bacterial Leaf Diseases. While halo blight (Pseudomonas coronafaciens) and bacterial stripe (P. striafaciens) were common in the early spring at the University Farm, Fayetteville, and apparently also at the Rice Branch Station, Stuttgart, there appeared to be at least one other bacterial species that was causing leaf blight even more abundantly than these two well known species. Since there was no opportunity for experimental study, the identity of this species is unknown. The symptoms briefly are as follows: Water-soaked green areas followed by grayish green and finally browning or blackening of tissues in the form of irregular blotches and spots without halos on blades and sheaths, and with the affected areas teeming with bacteria. Blades with one or more localized infections often turn red and symptoms are then likely to be confused with those of a relatively large number of other oat diseases.

The general appearance of the diseased areas reminded the writer of one of his earlier discoveries (A bacterial disease of foxtail. Annals Mo. Bot. Gard. 9: 333-402, 1922) recently revived by A. G. Johnson et al. (Bacterial blight and stalk rot of corn. Jour. Agr. Res. 78: 719-732, 1949), who found the same bacterium, originally isolated from a weed, now coming into prominence by attacking corn a quarter of a century later. This organism, Pseudomonas alboprecipitans, has the dangerous ability of attacking not only foxtail but almost all of the grains, including corn, sorghum, wheat, barley, rye, and oats, as Rosen and later Johnson found. So far as known, this disease up to the present has not been described on oats growing under natural conditions. However, the symptoms on oats in artificial inoculations by the foxtail organism are similar to those found on oats in the spring of 1950 at the University Farm and in oat material gathered by Dr. C. R. Adair at the Rice Experiment Station at about the same time. But the bacterial disease of foxtail and corn is typically a high temperature disease, while the one described here on oats was found in April and May with mean temperatures of 56° and 66° F. respectively.

Still another bacterial disease of oats, very briefly mentioned by the writer in his publication listed above (p. 338), needs to be considered, and if the disease on oats found in 1950 appears in such abundance in the future, it will doubtless receive more attention.

Helminthosporium Blight (H. sativum var.) (? H. victoriae), as in previous years was present in relatively minor proportions in the State as a whole, although in fields of susceptible Victoria derivatives grown in succession without rotation, there was a marked reduction in yield due primarily to this disease. However, such observed fields were few.

Smuts (Ustilago avenae and U. kolleri). In 1950 Arkansas growers continued their unenviable record of losing about 5 percent of their crop from smut, loss running as high as 20 percent in some fields. Outside of the rice growing area, where custom seed treatment is commonly used, seed treatment elsewhere appeared quite sporadic. County agents report that it is diffi-

cult to interest growers in treating seed with New Improved Ceresan in powder form because it makes them sick, and slurry machine treatment with Ceresan M does not always yield uniform results, some seed being overtreated and others not enough, aside from the fact that the machines now available are too expensive for most growers. It is hoped that replacement of New Improved Ceresan by Ceresan M, used either as a powder in a rotary drum or as a slurry without using expensive machines, or the use of Panogen, will overcome most of the objections to seed treatment.

Leaf Reddening and Red Spot Mosaic. There was much leaf reddening and gradual deterioration of stools and whole plants particularly in the spring of 1950. Much of this could be associated with aphid infestation, excessive soil moisture, or lack of nitrogen. In other instances no such association was found. In the nursery and in larger field plots at the University Farm, a number of varieties and selections showed typical symptoms of red spot mosaic (PDR 32: 172-175, 1948), the disease being systemic in that a characteristic reddish spotting which gradually turns brown occurs on every leaf of an affected plant although not nearly so abundantly on upper leaves. It is this systemic character plus the failure to isolate any recognizable causal microorganism which has led the writer to conjecture a virus origin. However, repeated attempts to produce the disease by mechanical transmission with juice from diseased plants have failed to produce systemic symptoms. Localized spots have been produced by using juice from diseased plants, when checks rubbed with carborundum and otherwise treated the same way, using the juice from healthy plants, failed to show spotting; but until systemic infections are produced artificially there is no good proof for the virus theory. Of course it is possible that oat red spot mosaic represents one of those viruses with which it is very difficult if not impossible to produce systemic symptoms at will although it is comparatively easy to obtain local lesions.

Whatever the cause of red spot mosaic may be, there can be little doubt that the disease was responsible for an appreciable loss in yield in the variety Traveler. All early fall-planted plots had approximately 30 to 40 percent of the plants showing this disease, in contrast to late planted ones in adjoining plots which had little or none. The difference in yields was impressive: with 9 replicates (1/50 acre per replicate) for each planting date, all the earliest planted plots, September 23, averaged 54.3 bushels per acre, those sown October 7 averaged 61.8 bushels, those sown October 21 averaged 85.2 bushels, and those sown November 4 averaged 89.4 bushels. Since there was very little Helminthosporium blight in any of the plots and no detected difference in amounts of any other disease, there is reason to assume that the marked difference in yield between the early-planted oats and those planted later was primarily due to the difference in amount of red spot mosaic. So far as the direct influence of weather is concerned, the usual experience has been that late-planted oats, being more subject to winter injury, usually yield less than those sown early.

It should be noted that the early-planted plots had more fall growth and greater aphid and leaf hopper infestations. There is also evidence indicating that aphids and leaf hoppers by themselves were not responsible for the marked difference in yields since varietal tests of both nursery and larger field plots, with all varieties planted at the same time and subject to the same amount of insect infestation, still showed differences in yield, these differences so far as Traveler (and several other varieties) are concerned being best explainable by the difference in amount of red spot mosaic. Thus, in the nursery Traveler yielded at the rate of 60.7 bushels per acre (9 replicates) and had considerable red spot mosaic, while R19-53-4-3 planted on the same date yielded 84.4 bushels and had no red spot mosaic. Both had heavy fall infestations of leaf hoppers and aphids.

Stem Rust (Puccinia graminis). There was very little stem rust on winter oats in 1950 but on spring oats it became quite common as the season advanced.

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STUDIES ON THE CONTROL OF GOLDEN NEMATODE OF POTATOES, WITH SYSTOX SPRAY (E-1059), AN ORGANIC PHOSPHATE INSECTICIDE

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Systox spray (E-1059), [1] an organic phosphate insecticide developed by Gerhard, Schrader of Farbenfabriken Bayer, Leverkusen, Germany, has been reported to penetrate plants by absorption, entering into seeds, roots or leaves and killing aphids, mites and some types of chewing insects. In view of this report preliminary tests with E-1059 were undertaken to determine its effectiveness in controlling the golden nematode of potatoes, Heterodera rostochiensis Wollenweber 1923, and root-knot nematodes (Meloidogyne sp.).

Systox spray is a brown viscous material containing 32.1 percent active ingredient with 67.9 percent emulsifier 8139 (active ingredient 3 lbs. per gallon). In the following experiments, all dilutions were made with water and concentrations used were based on percentage active ingredient.

TREATMENT OF GOLDEN NEMATODE CYSTS WITH E-1059: One gram samples of moist golden nematode cyst material (approximately 1500 cysts) were submerged in petri dishes containing E-1059 in concentrations of 0.25, 0.5, 1, and 2% for periods of one day and of one week, to determine the ability of E-1059 to penetrate the cysts, either through the wall or natural openings, and to affect larvae. After the treatment periods the cysts were washed thoroughly until no odor of the chemical could be detected. The treated cysts and similar lots of untreated cysts of the same origin were then placed in clean petri dishes and allowed to dry for two days, after which 25 cc of fresh potato leachings (collected by allowing water to seep through the soil of a vigorously growing potted potato plant) were added to stimulate hatching. The dishes were kept in the laboratory at room temperature. Daily examinations of the dishes were made with the aid of a binocular dissecting microscope and all hatched larvae removed and recorded. Results of this experiment are shown in Tables 1 and 2. The inhibition period indicated in the tables refers to the period between the adding of potato leachings and the appearance of the first larvae.

Table 1. Hatching of Heterodera rostochiensis larvae after exposure of cysts to E-1059 for one day and subsequently to potato leachings for 61 days.

E-1059 concentration (%)	Inhibition period (days)	Total hatched
Check	0	5780
0.25	10	586
0.5	10	275
1.0	33	9
2.0	61	0

Table 2. Hatching of Heterodera rostochiensis larvae after exposure of cysts to E-1059 for one week and subsequently to potato leachings for 55 days.

E-1059 concentration (%)	Inhibition period (days)	Total hatched
Check	0	6520
0.25	20	212
0.5	41	17
1.0	37	18
2.0	55	0

Several investigators (Carroll 1933; O'Brien, Gemmell, Prentice, and Wylie 1939; Smedley 1939; Feldmesser and Fassuliotis 1950) have found that exposure of cysts to various chemicals and treatments will inhibit larval hatching.

[1] Supplied by Geary Chemical Corporation, New York. Hereafter referred to as E-1059.

In these experiments, the inhibition period for cysts submerged in E-1059 for one day, ranged from ten days for the lowest concentration. Cysts treated for one week showed longer periods of inhibition and the total number of larvae which hatched after a one week treatment was less than the total number hatched from the cysts treated for one day. This was true for all concentrations. When the experiment was terminated, cysts from all concentrations taken from the dishes and broken open under the binocular dissecting microscope contained moving larvae, therefore, it is assumed that exposure to E-1059 disrupted, in some way, the stimulatory effect of potato root leachings to the larvae, or the response of the larvae to the stimulation.

A small experiment was then designed to find a concentration and period of treatment at which E-1059 would be lethal to larvae in cysts. Cysts submerged in concentrations of 2, 2.5, 3, 3.5 and 4% for periods of one day up to seven days were washed thoroughly and placed in petri dishes with 25 cc of potato leachings. After 30 days, cysts from all concentrations were opened and found to contain moving larvae. No larvae hatched during this period.

Both Hurst and Triffitt (1935) and Carroll and McMahon (1937) found that ferric oxide added to potato root leachings delayed hatching. In an effort to test the effect of E-1059 when added to leachings, an experiment was set up in which cysts were submerged in a mixture of equal parts of a .006% concentration of E-1059 and potato root leachings. Larval hatching was inhibited for 10 days at which time the experiment was terminated. Hatching occurred in the untreated checks within 24 hours.

TREATMENT OF GOLDEN NEMATODE LARVAE WITH E-1059: Newly hatched larvae of the golden nematode H. rostochiensis were exposed to E-1059 in petri dishes at concentrations of 0.25, 0.5, 1, and 2% for periods of one day and of one week to determine whether or not the compound was capable of killing them. After the exposure period the E-1059 solution was siphoned off carefully and replaced with tap water. Three weeks later counts were made with the aid of a binocular dissecting microscope and the number of dead and living larvae recorded. Table 3 shows the results of this experiment.

Table 3. Lethal properties of E-1059 when placed in direct contact with Heterodera rostochiensis larvae.

E-1059 Conc. (%)	Effect on larvae: After one day exposure: Number alive	Number dead	Percent killed	After one week exposure: Number alive	Number dead	Percent killed
Check	800	0	0	800	0	0
0.25	749	51	6.25	44	756	94.5
0.50	750	50	6.25	10	790	98.75
1.0	510	290	36.25	0	800	100
2.0	0	800	100	0	800	100

A low percentage of the larvae were killed when in direct contact with the material for one day, except at the highest concentration. The percentage kill for larvae exposed to the chemical for one week, however, was high for all concentrations tested.

Larvae appeared to be stunned or paralyzed when placed in the various concentrations of E-1059, but seemed to overcome this effect at the lower concentrations when the chemical was replaced with tap water. At the higher concentrations, E-1059 had a more definite injurious effect on the larvae, appearing under the microscope as constrictions in the gut wall. The disappearance of the line of demarcation between gut wall and outer cuticle was also evident. These malformations appeared after one or two days exposure to the material.

Hurst and Triffitt (1935) showed that a 2.5% solution of ferric chloride paralyzed hatched larvae in a few hours. Ferrous sulfate in strong solutions was lethal to these larvae but worked less rapidly. Smedley (1939) in testing larvae against the isothiocyanates, found that .02% ethyl isothiocyanate killed the larvae in two and one-half hours.

The fact that concentrations of 0.25% and 0.5% for one day killed only 6.25 percent of the larvae treated, while the same concentrations for one week killed 94.5 and 98.75 percent respectively, would indicate that the E-1059 is absorbed gradually and that death of the larvae is not sudden.

TREATMENT OF POTATO SEED PIECES: Potato seed pieces of approximately the same size and with only one cut surface which had been allowed to heal over were soaked in solutions of E-1059 at concentrations of .01, .03, 0.1, 0.25, 0.5, 1, and 2% for periods of one hour, two hours, and four hours. The seed pieces were then allowed to dry prior to planting in infested soil. Four-inch pots were used and each treatment was replicated three times. The plants were allowed to grow for a period of six weeks.

The soaking of potato seed pieces in solutions of E-1059 resulted in very poor germination with only about 60 percent of those planted showing any top growth. Tuber weights, taken from plants which showed somewhat normal growth, were inversely proportional to the concentrations of E-1059. The higher the concentration of E-1059 used, the lower the tuber weight.

In order to measure the effectiveness of the treatment, the number of "new" cysts (i.e., those which had developed on the roots of the plant sample recognizable by their color) was determined as number per gram of root. A modification of the method used by Chitwood and Feldmesser (1948) was used to separate the cyst from the soil and roots.

A reduction in the formation of new cysts was not evident.

TREATMENTS AS A FOLIAGE SPRAY: The purpose of this experiment was to determine if concentrations of E-1059 when sprayed upon the foliage would be absorbed and translocated to the roots in sufficient quantities to be lethal to the larvae of nematodes which infect the roots.

Potato seed pieces were planted in four-inch pots of golden nematode infested soil. Soon after the shoots emerged the plants were sprayed once weekly or twice weekly with E-1059 in concentrations of .006, .01, .05, 0.1, and 1%. Each treatment was replicated three times. The plants were sprayed for a period of four weeks, using a small atomizer spray[2]. Controls receiving no spray were included.

Phytotoxicity was very pronounced at the 1% concentration. Burning, slight curling of the leaf tips, and premature defoliation were the chief symptoms.

Examinations for cysts were made as in the previous experiment. The result of this experiment proved the treatments completely ineffective so far as any effect on the nematodes was concerned. Plants sprayed with all concentrations produced new cysts in numbers and stages of development comparable with those produced on the control.

TREATMENTS AS A SOIL DRENCH: An experiment was designed to determine whether or not E-1059 would inhibit hatching and be lethal to larvae in soil as it was in vitro. Four-inch pots of heavily infested soil were drenched with E-1059 in concentrations of .03, 0.25, 0.5, 1, and 2%. Each treatment was replicated six times, three pots receiving one application and three pots receiving a second application after one week. At the end of one week, the soil in the pots receiving one application was loosened and potato seed pieces were planted. The same procedure was followed one week later for those pots receiving two applications of E-1059.

In soil drenched with all the concentrations of E-1059, only 40 percent of the seed pieces germinated in soil receiving one application and 6 percent in soil receiving two applications. Top growth of those which did germinate was poor and tuber weights were inversely proportional to the concentration of the drench. No reduction in the number of new cysts formed was evident.

EXPERIMENTS ON ROOT-KNOT NEMATODE CONTROL: Since root-knot nematodes do not form cysts and the soft bodied females deposit their eggs in a mucoid mass, the eggs and larvae would appear to be more vulnerable than those of the golden nematode; so an experiment was set up to test the effect of E-1059 on root-knot nematodes when used as a soil drench.

Clean soil was placed in four-inch pots and each pot inoculated with 6 grams of root-knot inoculum. The pots were drenched with E-1059 solutions in concentrations of .03, 0.25, 0.5, 1 and 2%. After one week soil from each pot was mixed with enough additional nematode free soil to fill three pots. Tomato seedlings were planted in all pots. Inoculated controls which received no drench treatment were included.

At the higher concentrations, the chemical proved very phytotoxic, killing some of the tomato seedlings. Even at the lowest concentration, considerable injury was evident. Plants that did grow showed no signs of root-knot infection while the checks were severely galled.

In another experiment, spraying the foliage of tomato and tobacco seedlings growing in infested soil with concentrations of E-1059 up to 1% either once a week or twice a week did not control root knot. At the 1% concentration, burning and curling of the leaves of sprayed plants was observed.

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[2]DeVilbiss atomizer No. 15.

DISCUSSION AND CONCLUSIONS: Larval migration from golden nematode cysts treated in the laboratory was inhibited at all concentrations of E-1059 tested and hatched larvae were killed when exposed to various concentrations of E-1059 in vitro. Seed pieces soaked in various concentrations of E-1059 germinated poorly. The chemical was toxic to plants sprayed with a 1% concentration. Soil drench tests resulted in a low percentage of germination of potato seed pieces and poor top growth for seed pieces which did germinate. Nematode infection was not reduced in any of these experiments. It would appear, therefore, at least from these experiments, that E-1059 is of little or no value as a control for the golden nematode.

Soil drench tests with E-1059 for the control of root-knot nematodes resulted in good control with concentrations as low as .03% but tomato seedlings planted one week after treatment were injured.

Further experiments are planned in the hope of finding a practical method of eliminating this difficulty.

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DIVISION OF NEMATOLOGY

AN UNUSUALLY HEAVY DEVELOPMENT OF PEA ENATION MOSAIC ON 218 LINES OF PEA IN THE FIELD

W. T. Schroeder

During the 1950 growing season 171 foreign introductions and 47 domestic varieties and strains of pea (Pisum sativum) were planted in the field for seed increase and observations on reaction to any diseases that might occur. The number of plants of each lot depended upon the number of seeds available and the germination thereof. The rows of the various lines of pea were bordered on either side by solid rows of Innovation, a variety known to be especially susceptible to pea viruses.

Aside from a mild development of root rot, there occurred a striking development of a virus disease, resembling in symptoms that described by Osborn (1) as pea mosaic (pea virus 1) and later by Stubbs (3) and Pierce (2) as Pea Enation Mosaic. Susceptibility was complete in every line except one (Table 1). The enation character was common to all lines in the field, but some did not exhibit so severe a development of the other symptoms, such as vein-clearing, chlorotic and necrotic leaf spots, and bud distortion. The enation symptom was mildest on the following lines: Geneva No. 168, Geneva No. 169, Geneva No. 170, Bridger (#33819-Gallatin Valley), Late W. R. Alaska (#57A801-Gallatin Valley), Reg. W. R. Alaska (#55503-Gallatin Valley). Bonneville (#430-Rogers) and Horal exhibited the enation symptom but none of the other symptoms. Only one line, Geneva No. 168 (P. I. No. 140295), had any healthy appearing plants at the end of the season. Whether or not these were resistant or merely "escapes" remains to be determined, but this particular line did show a rather mild development of the disease on the susceptible plants. All other lines except the aforementioned showed the gamut of symptoms described for this disease. The development of aphis was not particularly heavy during the season, although they did make an early appearance and persisted throughout the season.

Table 1. Field reaction of various lines of pea to the pea enation mosaic virus, Geneva, New York.

Geneva No.	P. I. No.	Number plants	Number plants with virus	Number healthy
101	172338	59	59	0
102	172339	63	63	0
103	172340	62	62	0
104	172341	59	59	0
105	172342	24	24	0
106	164304	19	19	0
107	164346	37	37	0
108	164612	1	1	0
109	164614	78	78	0
110	164669	102	102	0
111	164690	48	48	0
112	164758	9	9	0
113	164766	49	49	0
114	164779	20	20	0
115	164787	67	67	0
116	164548	44	44	0
117	164837	92	92	0
118	164838	36	36	0
119	164853	13	13	0
120	164147	37	37	0
121	164148	15	15	0
122	164182	62	62	0
123	164396	73	73	0
124	164417	45	45	0
125	164426	34	34	0
126	164523	33	33	0
127	164523	40	40	0

Table 1. (Continued)

Geneva No.	P. I. No.	Number plants	Number plants with virus	Number healthy
128	163125	17	17	0
129	163126	63	63	0
130	163127	58	58	0
131	163128	15	15	0
132	163129	39	39	0
133	163130	25	25	0
134	163131	74	74	0
135	163132	15	15	0
136	163133	7	7	0
137	163134	29	29	0
139	165564	26	26	0
140	165577	39	39	0
141	169604	9	9	0
142	166129	19	19	0
143	169600	19	19	0
144	169608	13	13	0
147	169602	66	66	0
148	166117	41	41	0
150	166188	14	14	0
151	169610	76	76	0
152	165965	10	10	0
153	166082	57	57	0
154	169605	25	25	0
155	169609	26	26	0
156	169603	25	25	0
157	167250	2	2	0
158	169607	14	14	0
159	166159	60	60	0
160	166142	80	80	0
161	167253	9	9	0
162	167251	3	3	0
163	167273	58	58	0
164	165897	71	71	0
165	155308	0	0	0
167	140165	113	113	0
168	140295	301	297	4
169	140296	134	134	0
170	140297	185	185	0
171	140298	134	134	0
172	141966	97	97	0
173	142441	71	71	0
174	142775	301	301	0
175	142777	139	139	0
176	143483	52	52	0
177	143484	40	40	0
178	143486	219	219	0
188	185183	37	37	0
189	174920	4	4	0
190	177054	20	20	0
191	179020	2	2	0
192	179457	16	16	0
193	179458	10	10	0
194	181801	13	13	0
195	181958	3	3	0
196	183713	12	12	0
197	173929	4	4	0
198	173930	15	15	0
199	174319	9	9	0

Table 1. (Continued)

Geneva No.	P. I. No.	Number plants	Number plants with virus	Number healthy
200	174320	9	9	0
201	174321	12	12	0
202	174322	10	10	0
203	174917	19	19	0
204	174918	7	7	0
205	174919	44	44	0
206	174921	37	37	0
207	174922	26	26	0
208	174923	8	8	0
209	174924	22	22	0
210	174925	13	13	0
211	174926	11	11	0
212	175226	3	3	0
213	175227	4	4	0
214	175228	17	17	0
215	175229	24	24	0
216	175230	22	22	0
217	175231	9	9	0
218	175232	31	31	0
219	175233	10	10	0
220	176721	5	5	0
221	177056	10	10	0
222	179019	10	10	0
223	179449	29	29	0
224	179450	12	12	0
225	179451	15	15	0
226	179452	17	17	0
227	179453	2	2	0
228	179454	0	0	0
229	179456	4	4	0
230	179459	7	7	0
231	179460	3	3	0
232	179461	18	18	0
233	179721	4	4	0
234	179722	23	23	0
235	179723	41	41	0
236	179724	0	0	0
237	179969	40	40	0
238	179970	8	8	0
239	179971	23	23	0
240	179972	37	37	0
241	180327	7	7	0
242	180328	24	24	0
243	180329	44	44	0
244	180470	27	27	0
245	180471	27	27	0
246	180472	38	38	0
247	180868	19	19	0
248	181800	43	43	0
249	182985	18	18	0
250	183068	30	30	0
251	183140	1	1	0
252	183141	45	45	0
254	183334	5	5	0
255	183336	12	12	0
256	183413	70	70	0
257	183414	45	45	0
259	183446	68	68	0

Table 1. (Continued)

Geneva No.	P. I. No.	Number plants	Number plants with virus	Number healthy
260	183467	13	13	0
267	183714	11	11	0
268	183910	7	7	0
269	183944	52	52	0
270	183945	19	19	0
271	183946	37	37	0

Variety	Source	Number plants	Number plants with virus	Number healthy
Oneida #4433	Rogers	39	39	0
Alderman 420804	Gallatin Valley	32	32	-
Willets Wonder	---	58	58	0
Banner 4308	Rogers	14	14	0
Ranger 48727	Gallatin Valley	52	52	0
No. 326 H45946	Associated	16	16	0
Wisconsin Merit	Northrup King	40	40	0
Onward	Woodruff	30	30	0
Pride 418A/S-2	Northrup King	80	80	0
Commando	---	60	60	0
T. Laxton	---	44	44	0
Telephone DP #77118	Associated	28	28	0
Miracle #4512909	Associated	51	51	0
Lincoln	---	35	35	0
Dwarf Freezer (Green Canner) #4516	Rogers	33	33	0
Wando	---	83	83	0
Perfected Wales #422/5-8	Northrup King	69	69	0
Bonneville #4380	Rogers	79	79	0
Pride	---	99	99	0
Early Perf. #22711	Washburn-Wilson	47	47	0
Creole	---	39	39	0
Stratagem (M.R. 441292)	Associated	17	17	0
Improved Profusion	Rogers	104	104	0
Major H44732	Associated	48	48	0
Garden Pod Freezer #45167	Rogers	3	3	0
Cascade #34812	Gallatin Valley	141	141	0
W. R. Perf. #35803	Gallatin Valley	109	109	0
Bridger #33819	Gallatin Valley	96	96	0
Horal	---	85	85	0
W. R. Perf. #0-8202	Woodruff	42	42	0
Rocket #58710	Gallatin Valley	135	135	0
Pride #39709	Gallatin Valley	71	71	0
Late W. R. Alaska #57A801	Gallatin Valley	100	100	0
Loyalty #451211	Associated	51	51	0
W. R. Surprise #59711	Gallatin Valley	74	74	0
Reg. W. R. Alaska #55503	Gallatin Valley	204	204	0
Early Perfection #405A/S-6	Northrup King	35	35	0
Defiance	Woodruff	7	7	0
Miracle #1-85255	Woodruff	80	80	0

Table 1. (Continued)

Variety	Source	Number plants	Number plants with virus	Number healthy
Innovation	Woodruff	33	33	0
Hyalite #75801	---	66	66	0
Early Perfection #36805	Gallatin Valley	104	104	0
Wyola #77274	Associated	40	40	0
Pacemaker	---	37	37	0
Early Perf. #412	Rogers	62	62	0
Shasta #56005	Associated	22	22	0
T. Laxton #63A800	Gallatin Valley	63	63	0

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NEW YORK STATE AGRICULTURAL EXPERIMENT STATION, GENEVA, NEW YORK

FIELD REACTION OF 17 TOMATO VARIETIES TO THREE DISEASES IN 1949 AND 1950 AT GENEVA, NEW YORK

W. T. Schroeder

In 1949, 17 varieties of tomato were planted in random order in each of six blocks in a field known to be uniformly infested with the causal organism of Verticillium wilt (V. albo-atrum) of tomato. The same varieties were planted in 1950, but the blocks were planted crosswise to those of the previous year. The original intent to the planting was to determine the relative susceptibility of some commercial tomato varieties along with several supposedly Verticillium-tolerant lines of tomato. During the course of the test, other diseases appeared, particularly blossom-end rot and anthracnose, and data were also obtained on these diseases.

The varieties used and the data obtained are outlined in Table 1. There existed a highly significant difference among varieties with respect to blossom-end rot (non-parasitic) in both years. The fact that these differences were rather consistent and uniform for both years is indicated by the lack of significance between years or between varieties and years. This occurred in spite of the fact that the monthly mean temperatures for 1949 were above normal during the critical months for blossom-end rot development and below normal in 1950.

Anthracnose (Colletotrichum phomoides) development was significantly greater in 1950 than in 1949. All varieties except Stokescross #5, Rutgers, and Wisconsin 55 developed more anthracnose in 1950 than in 1949. These varieties are of later maturity than the others, and in all probability would have shown more anthracnose development had another harvest record been made, but lack of time prevented this. The discrepancy in these three varieties accounts in large part for the significant interaction between varieties and years.

The development of Verticillium wilt was significantly greater in 1950 than in 1949 although the difference is not too great. Highly significant differences among varieties occurred. Improved Wasatch Beauty, 46-31, Gem, John Baer, and Red Jacket are especially susceptible to Verticillium wilt; whereas, Stokesdale, Longred, and Wisconsin 55 are somewhat tolerant. While this degree of tolerance does not approach the resistance desired and found in some lines of tomato not adapted to New York State, it has some practical value in commercial plantings.

Table 1. Varietal reaction in the field of 17 tomato varieties to 3 diseases in 1949 and 1950, Geneva, New York.

Variety	Blossom-end rot Percent			Anthracnose Percent			Verticillium wilt Index[a]		
	1949	1950	Av.	1949	1950	Av.	1949	1950	Av.
Improved Wasatch Beauty	1.4	1.3	1.3	34.1	37.7	35.9	57	63	60
46-31	1.9	2.4	2.2	17.6	30.0	23.8	61	71	66
Early Jersey	2.6	8.6	5.6	40.6	39.8	40.2	45	46	46
Gem	2.1	3.6	2.8	27.8	39.2	33.5	52	58	55
Stokesdale	4.5	3.7	4.1	30.5	34.7	32.6	44	42	43
Stokescross #2	2.4	4.7	3.5	22.4	42.8	32.6	49	53	51
Stokescross #5	14.6	15.9	15.2	25.2	13.2	19.2	47	48	48
Valiant	2.9	5.4	4.2	40.8	55.0	47.9	46	51	48
John Baer	20.7	21.5	21.1	18.4	24.4	21.4	65	69	67
Red Jacket	14.8	10.6	12.7	13.8	31.3	22.6	55	68	62
Longred	12.8	14.8	13.9	21.6	30.8	26.2	44	41	42
Rutgers	27.8	15.7	21.8	24.0	13.3	18.7	49	50	50
Wisconsin 55	11.8	29.7	20.8	39.1	36.0	37.6	32	41	36
Geneva #6	18.1	13.5	15.8	20.8	26.5	23.7	50	44	47
Geneva #7	8.4	8.4	8.4	25.4	30.5	28.0	51	51	51
T-16	10.5	7.2	8.9	6.8	10.9	8.8	36	42	39
Club 86	6.8	14.2	10.5	7.8	7.8	7.8	48	49	48
L.S.D. (19:1)	7.4	8.3	5.5	8.7	26.0	13.6	7	7	5
(99:1)	9.8	11.0	7.3	11.6	34.5	18.0	9	9	6
Yearly Mean	9.7	10.7		24.5	29.6		49	52	

[a] Wilt Index: Based on a combined evaluation of the percentages of defoliation and leaf-spotting symptoms attributed to Verticillium wilt.

0 = healthy, no defoliation nor any sign of leaf-spotting;
20 = 1-25% defoliation, or leaf-spotting on up to 40% of foliage;
40 = 26-50% defoliation, or leaf-spotting on 41-60% of foliage;
60 = 51-75% defoliation or leaf-spotting on 61-80% of foliage;
80 = 76-95% defoliation, or leaf-spotting on 80-100% of foliage;
100 = plant practically dead except for a few tip leaves, 95-100% defoliation.

NEW YORK STATE AGRICULTURAL EXPERIMENT STATION, GENEVA, NEW YORK

SCLEROTINIA STEM ROT AND RAMULARIA LEAF SPOT OF DANDELION GROWN FOR SEED IN SKAGIT COUNTY, WASHINGTON

D. M. McLean

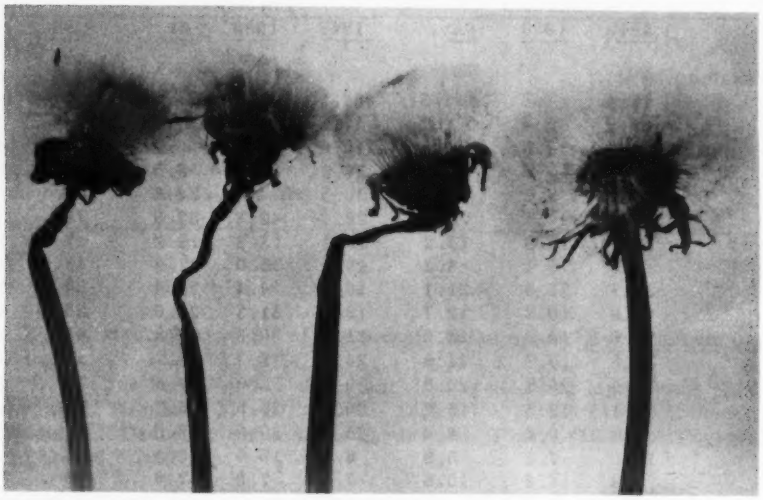

FIGURE 1. Dandelion seed head and peduncle rot caused by Sclerotinia sclerotiorum. Healthy seed head and peduncle at right.

FIGURE 2. Ramularia leaf spot on cultivated dandelion.

The Puget sound area, and particularly the Skagit Valley in Northwest Washington, is renowned for its production of vegetable seeds. The area is particularly adapted for production of cruciferous seeds. A very limited acreage of dandelion (Taraxacum officinale Weber) var. Arlington Broadleaf is grown for seed as a specialty crop in the vicinity of Mount Vernon, Washington. An unusual peduncle rot caused a considerable loss of seed in the 1950 crop (Figure 1). The disease was determined to be caused by Sclerotinia sclerotiorum (Lib.) Dby. This fungus is a common pathogen, often causing serious losses in cruciferous seed crops. The prevalence of the disease on dandelion only adds to the hosts perpetuating the organism in this concentrated seed-producing area.

Primary infections occur on the bracts subtending the head, probably originating on the fallen flower petals. The fungus invades the receptacle and grows very rapidly down the succulent peduncle, eventually causing decay of the affected tissue before the seeds are mature. Sclerotia have not been observed on the decayed peduncle tissues although it is very likely that they occur on the infected flower heads which fall to the ground.

A common leaf spot caused by Ramularia taraxaci Karst. [1] is found both on cultivated (Figure 2) and on wild dandelion.

COOPERATIVE INVESTIGATIONS OF THE DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, THE NORTHWESTERN WASHINGTON AGRICULTURAL EXPERIMENT STATION, MOUNT VERNON, WASHINGTON AND THE STATE COLLEGE OF WASHINGTON, PULLMAN, WASHINGTON

[1] Determined by Charles Gardner Shaw, Department of Plant Pathology, Washington State College.

BROWN SPOT DISEASE OF CELERY FOUND IN NEW YORK

Ralph Segall

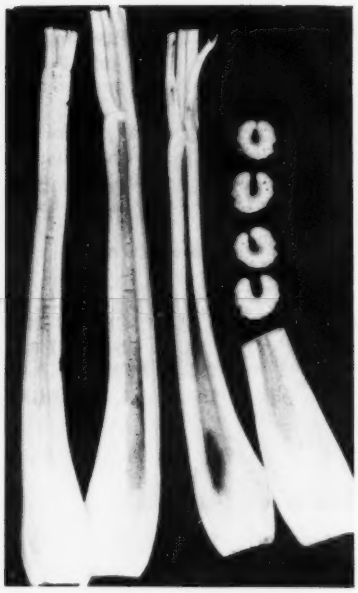

FIGURE 1. Stalks of green pascal celery showing shallow brown discoloration of the inner surface of the petiole and adaxial cracks in which the causal organism Cephalosporium sp. fruited and formed spores.

New strains of green pascal celery grown on muck soil in Orange County, New York were found with a brown discoloration on the inside surface of the petiole in July 1949. A fungus was isolated from the infected tissue and identified as Cephalosporium. When this isolate was reinoculated onto green pascal and self blanching celery varieties by means of spore suspensions, typical symptoms resulted. Smith and Ramsey (1) reported a brown spot disease caused by Cephalosporium sp. of celery grown in Colorado, characterized by brown discolored areas on the under surface of the stalk. Optimum growth of this fungus in culture was found to be 75° F. The 1949 growing season in Orange County, New York was characterized by high summer temperature. The average monthly temperatures were as follows: June -- 71.5° F., 4.0° above normal; July -- 76.5° F., 4.6° above normal; and August -- 74.0° F., 4.2° above normal. These average temperatures coincided rather closely with the optimum growth temperature of the fungus, found by Smith and Ramsey to be 75° F.

During the 1950 season, a careful check was made for the disease in Orange County on the same fields but none was found. The temperature during the 1950 season was slightly below normal with the following average monthly temperature: June -- 66.3° F., 1.2° below normal; July -- 70.2° F., 1.7° below normal; August -- 69.6° F., 0.2° below normal. The fact that the brown spot disease of celery coincided with a season during which temperatures were unusually high might account for its presence in 1949 and not in other years.

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DEPARTMENT OF PLANT PATHOLOGY, CORNELL UNIVERSITY, ITHACA, NEW YORK

VERTICILLIUM WILT OF COTTON IN ALABAMA

Albert L. Smith and Coyt Wilson

Beginning in 1947 observations have been made on Verticillium wilt (V. albo-atrum) of upland cotton (Gossypium hirsutum) in Alabama. The disease was first observed in a planting of Empire variety on the Tennessee Valley Substation near Belle Mina in September, 1947. This field had been planted continuously to cotton since 1928 when the station was established and probably had been in continuous cotton prior to 1928. This original spot involved approximately one-half acre and only scattered plants were diseased. As far as the writers can determine this is the first observation on Verticillium wilt of cotton in Alabama although the organism was recovered by Carpenter (1) in 1918 from okra plants grown in the vicinity of Birmingham.

Subsequently the disease has been observed in several fields in the vicinity of Decatur, in an area involving two counties. The disease to date has been confined to the Decatur soil series. The average pH of the surface soil of the Decatur series is 5.5 and the average for the subsoil is 5.4. The range in pH is from 5.0 to 6.3. The Decatur series is characterized as a clay loam, brownish-red in color, and relatively high in exchangeable calcium content.

Losses from this disease have been quite mild. In most cases only scattered plants were affected. Well defined infested spots are established in some locations in which the disease may appear one year and entirely disappear the next, depending largely on moisture and temperature in July and August. Only one report has been received indicating general loss to a crop of several acres.

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ALABAMA AGRICULTURAL EXPERIMENT STATION IN COOPERATION WITH THE DIVISION OF COTTON AND OTHER FIBER CROPS AND DISEASES, U. S. DEPARTMENT OF AGRICULTURE, AUBURN, ALABAMA

DISEASES OF GUAR IN GEORGIA [1]

E. S. Luttrell

During the summer of 1950, plantings of guar (Cyamopsis tetragonoloba (L.) Taub.) in the Southeastern Regional Plant Introduction Nursery located at the Georgia Experiment Station were severely damaged by two diseases, white mold (Sclerotium rolfsii Sacc.) and Alternaria leaf spot (Alternaria cucumerina (Ell. & Ev.) Elliott ?). Observations indicate that both of these diseases may prove to be important factors in limiting the production of guar in Georgia as a green manure crop, or especially, as a seed crop. None of the 55 strains of guar in this planting, all of which were recent introductions from India, showed a satisfactory degree of resistance to either disease.

White mold was present throughout the summer and affected plants of all ages. It caused moderate reductions in stands of all strains. In view of the extremely dry season, the incidence of infection was surprisingly high. The most conspicuous feature of the disease was the presence of the heavy, white mycelial mats of Sclerotium rolfsii surrounding the stem at the ground level. Following the appearance of the mycelium, the entire shoot of the plant quickly wilted and died. On dying plants the surface mycelium of the fungus produced its characteristic spherical, brown sclerotia. The roots of diseased plants were rotted, and the bark under the mycelial mat was shredded and sloughed off of the roots and base of the stem.

Although no observations were made on seedlings, Alternaria leaf spot was abundantly present on older plants throughout the summer and fall up to the time of killing frosts. By the end of the summer almost 100 percent of the leaves were infected and considerable defoliation occurred on all strains of guar. Some variations in the amount of damage caused by the disease were observed among these strains, but they appeared to be the result of differences in degree of maturity rather than susceptibility. One strain, No. 164476, was completely killed out before the pods matured. However, this was an early maturing strain, and the heavy damage it suffered may be attributed to the fact that it matured in August when the disease was at its peak. The less severely infected strains had not yet bloomed at this time and probably for this reason were in better condition to tolerate infection.

The typical leaf spots were roughly circular and measured 3 to 6 mm. in diameter. They were tan to brown and were surrounded by a narrow, dark-brown border which was entire or irregularly dentate. Concentric ridges in the dead leaf tissue gave the spots a zonate appearance. In moist weather both surfaces of the spots were covered with a dark-brown felt of conidiophores. In addition to the larger spots the leaves were heavily marked with small, angular, dark-brown spots. These spots were often confluent to form an irregular patchwork. Although this disease was primarily a leaf spot, it also affected the stems and pods. On the stems it produced black flecks and oval to elliptic spots 0.5 to 2 mm. long. The lesions were slightly depressed to raised and were often confluent to produce a black discoloration over areas of the stem. The pods were covered with short, black streaks obliquely transverse to its longitudinal axis. The streaks became confluent to form black patches. Some of the seed in infected pods were shriveled and blackened and covered with a white mycelium.

The conidiophores of the causal fungus formed an almost continuous layer over the surface of the lesions. They were erect, simple, cylindrical, straight, marked with frequent conidial scars, and medium brown to fuliginous to subhyaline at the tips. They measured 42-78 x 5.6-7 microns. The conidia were obclavate and fuliginous to ultimately dark brown. They were divided by 3 to 7 primary transverse septa and were constricted at the septa. Most of the cells were provided with 1 to 3 longitudinal septa and often with secondary transverse and oblique septa. The base of the conidium was rounded and provided with an inconspicuous circular hilum. The apical end terminated in a long, delicate, hyaline process which was often longer than the body of the conidium. This structure was 0 to 2-septate and 2.2-2.8 microns in diameter. The conidia, including the apical process, measured 81-199 x 13.4 - 23.8 microns. This fungus was isolated from conidia and from leaf, stem, and pod lesions. Morphologically it closely resembles Alternaria cucumerina. It may be tentatively referred to this species or at least to the A. cucumerina group.

Conidia of Alternaria cucumerina were rarely found on dead stems. Such stems were usually covered with other, presumably saprophytic, fungi. The most conspicuous of these were a species of Cercospora, which according to a communication from Dr. Charles Chupp may possibly belong under C. psoraleae Ray, an Alternaria species of the A. tenuis group, and a Fusarium species.

GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA

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[1] Paper No. 208, Journal Series, Georgia Experiment Station.

A LEAF SPOT OF SOYBEAN CAUSED BY SCLEROTIUM ROLFSII

S. G. Lehman, Harry Murakishi and Joe H. Graham

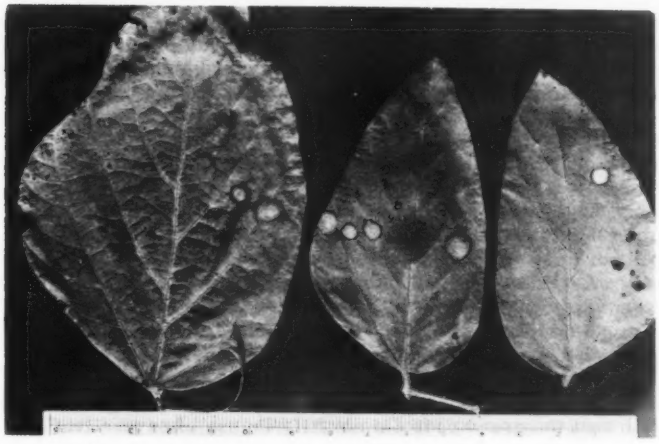

FIGURE 1. Lesions of S. rolfsii on soybean leaves taken from the field. Photo by Bernard Ellison.

Sclerotium rolfsii has long been known as a parasite of soybean and numerous other cultivated and wild species of plants. In the experience of the writers, parasitic attack by this fungus has always been limited to the stem at or near the soil level, to roots beneath the soil or to fruits such as cantaloupes, cucumbers, and cotton bolls, lying on the soil. The appearance of S. rolfsii in the role of a leaf spotting fungus occurs very infrequently and so far as the writers are aware has not been reported in the literature.

In late September, 1944, while examing soybeans on the Upper Coastal Plains Test Farm at Rocky Mount, North Carolina, where a high proportion of the plants in a large area were being killed by S. rolfsii, a considerable number of leaves were found with spots of diseased tissue differing in appearance from any previously observed on soybean leaves. The lesions were circular in outline and medium brown to light brown or straw color with a narrow band of darker necrotic tissue at the border, this narrow border being more conspicuous on the smaller than on the larger spots. Concentric circular markings were visible on many of the lesions (Figure 1). The diseased areas varied in size up to 1.5 cm., most of them being 1 cm. or less in diameter. At the center of many of the diseased areas a small clump of white mycelium was plainly visible. A small spherical brown sclerotium replaced this clump of mycelium on many of the lesions. In size and color these sclerotia resembled those of S. rolfsii occurring on the stems of other diseased plants in the area.

Some of the plants were leaning over so that their leaves touched the soil. Lesions were on leaves touching the soil, and on others 10 to 12 inches above the soil. Many of the infected leaves showed unmistakable evidence of soil splashed onto them by a heavy rain which fell 8 and 9 days previous to the date when the disease was first observed.

A fungus grew from sclerotia that had been taken from the leaves and planted on non-nutrient agar. When transferred to potato dextrose agar in tubes and petri dishes, this fungus produced numerous sclerotia in all essential details like those produced by cultures of S. rolfsii isolated from soybean stems.

Isolates from the leaves were tested for pathogenicity by placing bits of mycelium on leaves of soybean plants growing in the greenhouse. The inoculated plants were kept in an incubation chamber with cloth sides which were kept moist by water seeping from a canvas hose. After four or five days, infection became evident on the inoculated leaves as water-soaked spots which later took on much the same appearance as the spots seen on leaves in the field. Isolations from these diseased, greenhouse-grown leaves yielded cultures of S. rolfsii like the one used for inoculation.

Mycelium and sclerotia of the fungus isolated from diseased leaves were planted just beneath the soil against the stems of young soybean plants growing in pots in the greenhouse.

In the course of about three weeks after inoculation two-thirds of the inoculated plants died and produced sclerotia on the lower part of the stems. Other inoculated plants succumbed later. All control plants remained healthy to the end of the experiment.

In August 1950, a few lesions resembling those seen in 1944 were again found on soybean leaves at Rocky Mount in a field where some of the soybeans were being killed by S. rolfsii. A vigorous culture of S. rolfsii was obtained by plating one of these lesions on water agar. A mycelium suspension of this isolate sprayed on soybean leaves in the greenhouse produced large lesions after several days. Apparently, after splashing rain followed by a more or less prolonged period of high humidity, S. rolfsii may become a foliage parasite of soybean.

UNIVERSITY OF NORTH CAROLINA, RALEIGH

1950 PLANT DISEASE SURVEY FOR NEW MEXICO

Philip J. Leyendecker, Jr.

Heavy rains during July over much of the State resulted in crop damage caused by a number of unusual diseases which are not ordinarily of economic importance.

1. Rust (Uromyces phaseoli typica Arth.) on pinto beans in the irrigated regions around Estancia accounted for considerable damage this year. Periods of high humidity increased the severity of the rust in that the green pods did not fill because of severe leaf and pod infection. Airplane dusting with sulfur did not check the disease since the fungicide was applied after most of the damage had occurred and the pathogen was in the black spore stage. A total of eight to ten thousand acres were infected, and losses in yield were 10 to 25 percent.

2. Common Blight (Xanthomonas phaseoli (EFS) Dowson) was also prevalent throughout the bean producing areas of the State. Commercial fungicides were used by numerous farmers with negative results. Yields in severely infected fields were estimated to have been reduced 40 to 50 percent by the blight organism.

3. A virus disease of pepper (hot type) and tomato (Bonny Best) which had been reported as a possibly new disease in 1949 (P.D.R. 34: 39-40. 1950) has been identified as big bud. [1] The disease was again present in the Mesilla Valley on both crops and appeared to be increasing at an alarming rate. Infected tomato and pepper plants set no fruit and served as hosts for insect vectors before the disease could be recognized and the plants rogued from the field.

4. Wilt [2] of pepper (hot type), which can usually be economically controlled by cultural methods, caused considerable yield reductions that ranged from 20 to 100 percent in the Mesilla Valley. In spite of the use of the recommended high single-row seed beds, the soil moisture and temperatures promoted disease expression in July and August. Invariably, fields which had been irrigated before the heavy July rains were completely destroyed.

5. Stem and pod rot (Sclerotium rolfsii Sacc.) of peanut (Spanish and Arkansas Red) accounted for a 20 to 30 percent reduction in yield in the Portales area. Approximately 7,000 acres were involved accounting for a cash loss to the growers of about $200,000. The stem and pod rot was aggravated by high soil moisture and cool soil temperatures during July. In a number of fields, especially in the fields that had supported peanuts for four or five years, the entire crop was destroyed. The disease, however, was also present in previously cultivated fields planted to peanuts for the first time.

6. Net Blotch (Pyrenophora teres (Died.) Drechs.) of winter barley was found for the first time in the State in a field near Artesia. Most of the plants in a five-acre field appeared to be diseased. Tip necrosis of the younger leaves was pronounced while the older leaves were completely destroyed. This disease may have occurred before in the State, but this collection is the first authenticated identification.

7. Leaf Rust (Puccinia rubigo-vera tritici (Eriks.) Carleton) of wheat was very common this year in fall plantings. If climatic conditions promote disease expression in the spring of 1951, considerable damage can be expected. Many fields showed considerable leaf destruction early in the fall, and if mild weather prevails, the disease may be expected to appear in epiphytotic form.

8. Black Rot (Xanthomonas campestris (Pammel) S.A.B.) of cabbage was found causing some damage in the Cloudcroft area. In one field, the adjacent rows of turnip and radish also appeared

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[1] Dana, Oregon State College: Kendrick, University of California.

[2] Causal organism somewhat in question at present, will probably be classified by California workers in the near future.

to be infected.

9. Black Rot (Ceratostomella fimbriata (Ell. & Hals.) Ell.) of sweet potato was present in a number of plantings in the Mesilla Valley. Losses in some fields ran as high as 15 recent of the crop. Favorable soil moisture conditions during July probably accounted for the high disease incidence. Maryland is the most popular variety planted.

10. Scurf (Monilochaetes infuscans Ell. & Hals. ex Harter) of sweet potato was present in varying degrees in most plantings of sweet potatoes in the State. Market grade was reduced and some fields were not harvested because of the severity of the disease. Roots from fields which had been planted to sweet potatoes for three or four years were almost 100 percent infected.

11. Cotton Wilt (Verticillium albo-atrum Reinke & Berth.) was unusually severe during the 1950 season in the Mesilla Valley of New Mexico and in the adjacent El Paso Valley of Texas. Over 95 percent of the acreage of American Upland cotton is planted to strains of Acala. Losses from the Verticillium wilt of cotton vary from year to year depending upon the time of the season when soil temperatures become favorable for disease expression. Reductions in yield were very marked in the 1950 season because of the early appearance of the disease in mid-July. Ordinarily, in the Mesilla Valley, severe wilt symptoms appear in mid-August or later, and losses under such conditions are not as pronounced. In the 1950 season, reductions in yield ranged from 0 to 75 percent. It has been estimated that the overall loss in yield due to Verticillium wilt in the Mesilla Valley was approximately 1/6 of a bale per acre.

Increased acreages of American Egyptian cottons (SXP, Amsak and Pima 32) permitted more detailed observations of the effect of Verticillium on these purported highly tolerant strains. Typical leaf mottling and internal stem discoloration were evident throughout the season. Under New Mexico conditions, it appeared that the American Egyptian strains "outgrew" the disease. In most cases the early discoloration in the center of the stem was completely surrounded by healthy tissue formed later in the season. The American Egyptian varieties seemed to produce normal yields on land which was severely infested with the wilt organism.

11a. Verticillium albo-atrum was isolated from the following host plants during the season:

a. Xanthium commune Britton.
b. Sisymbrium irio (L.) Britton. (New host). Eliminates this plant as a potential winter green manure crop in cotton culture.
c. Hibiscus esculentus L.

12. Bacterial Blight (Xanthomonas malvacearum (EFS) Dowson) of cotton appeared again this past season in epiphytotic form in the Pecos Valley of New Mexico. Driving rains early in the season probably contributed to the dissemination and establishment of the blight organism from early centers of infection. Although most of the seed planted in the area was acid-delinted and Ceresan-treated, there was sufficient seedling infection to afford substantial inoculum for establishment of the disease.

By mid-season, the lower eight or ten leaves of the cotton plants were completely destroyed in entire fields. Subsequently, development of the black arm and boll rot stages of the disease became evident. Many fields suffered from early leaf and square damage which tended to make for a later crop. Damage caused by the disease in reducing yield and fiber grade cannot be accurately estimated, although it is known to be considerable. The seriousness of the blight problem is demonstrated by the fact that many growers are importing southeastern cottons into this area in a search for more tolerance to the disease than is present in the 1517 Acalas.

The extreme susceptibility of the American Egyptian strains to blight was also observed. In one ten-acre field of long staple cotton near Artesia, not a single normal boll was harvested. The few bolls that were set were completely over-run by the blight oragnism.

DEPARTMENT OF BOTANY, NEW MEXICO AGRICULTURAL EXPERIMENT STATION, STATE COLLEGE, NEW MEXICO

A LIST OF SOME OF THE PLANT DISEASES OCCURRING IN EVERGLADES REGION OF FLORIDA DURING THE 1949-1950 SEASON

Warren N. Stoner

The following plant disease list includes those which caused major economic losses from July 1949 to June 1950, at the locations indicated. The Florida State Extension Service in Broward, Collier, Hendry, Martin, Okeechobee, Palm Beach and St. Lucie counties has cooperated fully in this survey. Special thanks are due to agents M. U. Mounts, John Causey, H. L. Speer, L. M. Johnson, B. E. Lawton, Robert S. Pryor, H. L. Johnson and Charles D. Kime for their help and continued interest in the compilation of this report.

CROP Disease	Cause	Location
	VEGETABLE CROP DISEASES	
BEET		
Leaf spot	Cercospora beticola Sacc.	Belle Glade
BROCCOLI		
Downy mildew	Peronospora parasitica (Pers.) Tul.	Canal Point Belle Glade Okeelanta
CAULIFLOWER		
Downy mildew	Peronospora parasitica (Pers.) Tul.	Pahokee
CELERY		
Early blight	Cercospora apii Fres.	Everglades Expt. Sta.
Damp off	Rhizoctonia solani Kühn	Okeelanta Belle Glade
CUCUMBER		
Cucumber mosaic	Marmor cucumeris var. commelinae Holmes	Indiantown Loxahatchee Canal Point
Powdery mildew	Erysiphe cichoracearum DC.	Boynton Beach Fort Pierce
EGGPLANT		
Tip over	Phomopsis vexans (Sacc. & Syd.) Hart.	Bean City Canal Point Krammer Island
Wilt	Verticillium albo-atrum Reinke & Berth.	Loxahatchee Canal Point Indiantown Boynton Beach
HORSE-RADISH		
Powdery mildew	Erysiphe polygoni DC.	Belle Glade
IRISH POTATO		
Early blight	Alternaria solani (Ell. & Mart.) Jones & Grout	Belle Glade
Late blight	Phytophthora infestans (Mont.) DeBy.	Belle Glade Canal Point
Scab	Actinomyces scabies (Thax.) Güssow	Canal Point Belle Glade
LETTUCE		
Bacterial soft rot	Erwinia carotovora (L.R. Jones) Holland	Everglades Expt. Sta.
LIMA BEAN		
Brown stem	Rhizoctonia solani Kühn	Route 7 near Boynton Beach
PEPPER		
"Mosaic"	Marmor cucumeris var. commelinae Holmes	Indiantown

CROP Disease	Cause	Location
VEGETABLE CROP DISEASES (Continued)		
RADISH		
White rust	Albugo candida (Pers.) Rous.	Belle Glade
ROMAINE		
Lettuce mosaic	Marmor lactucae Holmes	Near Six-Mile Bend, Belle Glade
SNAP BEAN		
Cottony rot	Sclerotinia sclerotiorum (Lib.) Mass.	Everglades Expt. Sta.
Rust	Uromyces phaseoli var. typica Arthur	Boynton Beach
Bean mosaic	Marmor phaseoli Holmes	In all areas where beans grow
Powdery mildew	Erysiphe polygoni DC.	Belle Glade
Angular leaf spot	Isariopsis griseola Sacc.	Belle Glade
Brown stem	Rhizoctonia solani Kühn	Widespread in area
TOMATO		
Late blight	Phytophthora infestans (Mont.) DeBy.	Loxahatchee Fort Pierce Boynton Beach Indiantown
Gray mold	Botrytis cinerea Fr.	Fort Pierce Indiantown Boynton Beach Loxahatchee Delray Beach
Gray leaf spot	Stemphylium solani Weber	Bean City Fort Pierce Loxahatchee Boynton Beach
WATERMELON		
Anthracnose	Colletotrichum lagenarium (Pass.) Ell. & Hals.	Fort Pierce
Downy mildew	Pseudoperonospora cubensis (Berk. & Curt.) Rostow	Indiantown
FIELD CROP DISEASES		
ALFALFA		
Leaf spot	Pseudopeziza medicaginis (Lib.) Sacc.	Hillsboro Plantation
CORN		
Rust	Puccinia sorghi Schw.	Pahokee Everglades Expt. Sta. Boynton Beach
Smut	Ustilago zeae (Beckm.) Ung. (U. maydis)	Canal Point Boynton Beach Okeelanta
Leaf blight	Helminthosporium turcicum Pass.	Widespread in area Boynton Beach Fort Pierce
Leaf spot	Helminthosporium maydis Nishik. & Miy.	Belle Glade Fort Pierce
RICE		
Leaf blight	Cochliobolus miyabeanus Ito & Kuribay.	Okeelanta area Belle Glade

CROP Disease	Cause	Location
	SMALL FRUIT DISEASES	
STRAWBERRY Leaf spot	Mycosphaerella fragariae (Tul.) Lindau	Belle Glade
	ORNAMENTAL DISEASES	
GLADIOLUS Corm rot	Fusarium orthoceras Woll. var. gladioli McCull.	Delray Beach Belle Glade
Pansy "Mosaic"	Marmor cucumeris var. commelinae Holmes	Everglades Expt. Sta. Belle Glade
SPIDER LILY Pink or bloody spot	Serratia marcescens Bizio.	Canal Point
TRANSVAAL DAISY Leaf spot	Ascochyta gerberae	Pahokee

EVERGLADES EXPERIMENT STATION, BELLE GLADE, FLORIDA

NEW OR UNUSUAL RECORDS OF PLANT DISEASES

THE OCCURRENCE OF THE SUGAR-BEET NEMATODE IN ARIZONA

By Harold W. Reynolds

The sugar-beet nematode, Heterodera schachtii Schmidt, was found in November 1950, for the first time in Arizona. This pest is rather generally distributed in one 34-acre field, with one to two acres of the field having small stunted sugar beets which showed heavy infection. The field is located in the Salt River Valley where approximately 1000 acres of sugar beets are grown annually for the production of seed. This pest was not encountered in a survey made in the other 30 sugar beet fields of the Valley.

DIVISION OF NEMATOLOGY, U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, SACATON, ARIZONA

FUSARIUM WILT OF SESAME AT CHARLOTTE COURT HOUSE, VIRIGINA, IN 1950

By R. D. Sears and S. A. Wingard

Fusarium wilt of sesame was found at Charlotte Court House, Virginia, in 1950.

Plot	Variety	Percentage of Fusarium wilt	Plot	Variety	Percentage of Fusarium wilt
1	K 8	1	14	N 68 - 1	2
2	K 9	1	15	N 124-9-1--1	1
3	K 10	1	16	N 406-61--3-12	0
4	Y 3	2	17	N 413	1
5	Y 6	2	18	N 1025-3	1
6	Y 7	2	19	N 1032-1-1	1
7	Early Russian	0	20	N 1037	1
8	SC 4516	3	21	N 1040	1
9	SC 4520	1	22	N 119-3	0
10	SC 4521	2	23	N 2143	0
11	SC 4522	3	24	N 2345	3
12	Venezuela 51	1	25	N 2346	0
13	N 57	1			

Letters indicate State from which seed was originally obtained:
K-Kansas; Y-Arizona; SC-South Carolina; N-Nebraska

0 --- No wilt observed
1 --- 0 to 5% wilt
2 --- 5 to 10% wilt
3 --- 10 to 15% wilt

VIRGINIA AGRICULTURAL EXPERIMENT STATION, BLACKSBURG

ISOLATION OF THE OAK WILT FUNGUS FROM SWAMP WHITE OAK

By J. C. Carter and Noel B. Wysong

The oak wilt fungus Chalara quercina Henry was isolated from branch samples of a wilting swamp white oak (Quercus bicolor Willd.). These samples collected on July 8, 1950, were obtained from a tree twelve inches in trunk diameter, located in the Dan Ryan Woods of the Forest Preserve District of Cook County, Illinois. In 1949, this tree showed early symptoms of wilt when leaves on the tips of many scattered branches wilted, and many shoot tips died. By July 8, 1950, the tree showed some staghead and approximately half of the living portion of the crown showed leaf wilt. The wilting leaves were light brown or straw colored. The cambial region of

the branch samples was brown, but brown to black streaks were not evident in the sapwood.

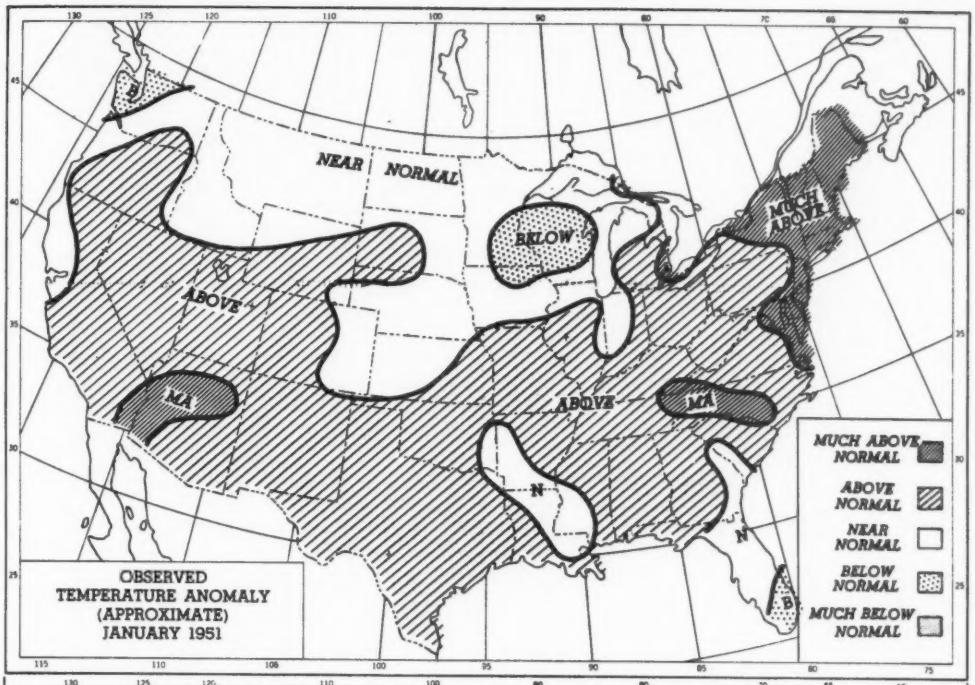
Infection of swamp white oak with Chalara quercina Henry by inoculation of plants grown under greenhouse conditions has been reported by Young [1]. However, it is believed that this is the first report of Chalara quercina Henry having been isolated from a naturally infected swamp white oak growing in the woods.

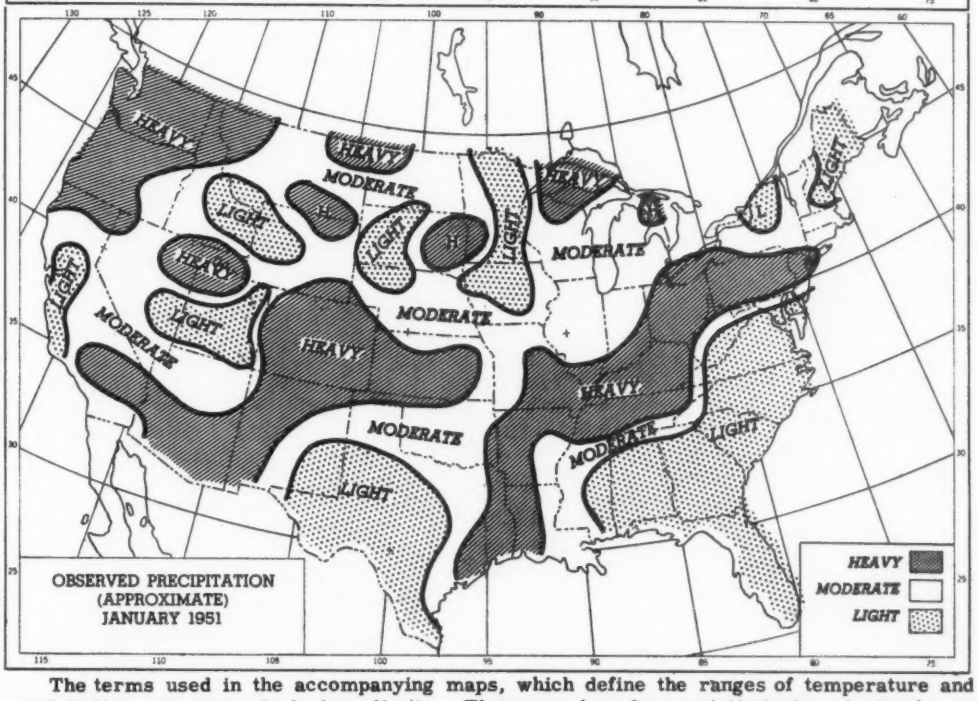
ILLINOIS STATE NATURAL HISTORY SURVEY, URBANA

A CORRECTION

F. P. McWhorter calls attention to a typographical error in the article by McWhorter and Anderson in the February 15 issue. On page 106, paragraph 2, line 6, reads "are not present in four," etc. The statement should read "are now present" etc.

[1] Young, Roy A. Studies on oak wilt, caused by Chalara quercina. Phytopathology 39: 425-441. June, 1949.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

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